

## 1. INTRODUCTION AND BACKGROUND

The Air Resources Board (ARB) prepared this report to the Legislature on indoor air quality in response to requirements of Assembly Bill 1173 (Keeley, 2002; Health and Safety Code Section 39930; see Appendix I). As required by the legislation, this report summarizes the best scientific information available on indoor air pollution, including:

- Common indoor pollutants and their sources.
- The potential health impacts of indoor pollutants and associated costs.
- Existing regulations and current industry practices.
- Options for mitigation in schools, non-industrial workplaces, homes, and other indoor locations.
- State and federal efforts related to control of biological and radiological substances.

In preparing the report, the ARB consulted with the Department of Health Services (DHS), the Office of Environmental Health Hazard Assessment (OEHHA), the California Energy Commission (CEC), the Department of Industrial Relations (DIR, Cal/OSHA), relevant industries and other stakeholders, and interested members of the public. On April 4, 2003 a public workshop was conducted to inform stakeholders about the report and the anticipated schedule, and to solicit comments from them. On June 30, 2004, another workshop was held to receive comments on the draft report. A website and an email list serve were established to keep stakeholders informed of progress on the report preparation. The website is available at <http://www.arb.ca.gov/research/indoor/ab1173/ab1173.htm>, which also includes information on how to sign up for the email list serve. ARB released a revised draft report in November 2004 for review by interested stakeholders and a University of California scientific peer review panel. A subsequently revised draft report is now considered by ARB Board members before being forwarded to the Legislature.

This report reflects key points from the large body of knowledge that has been generated about indoor air quality since it became a concern in the 1970s. Researchers worldwide have made great progress in identifying indoor pollutants and understanding their relationship to human exposure and resultant health effects. Several international conferences are now held annually, and one journal, *Indoor Air*, is devoted exclusively to the field of indoor air quality. Many additional journals carry articles on indoor air quality. This report summarizes key findings of the most recent literature, with emphasis on the major trends identified by multiple investigators.

### 1.1 INDOOR POLLUTION POSES A SIGNIFICANT HEALTH RISK

State and federal comparative risk projects have repeatedly ranked indoor pollutants and sources in the high-risk categories of their analyses relative to other environmental health problems. In the 1994 *California Comparative Risk Project* (CCRP, 1994), the Human Health Committee ranked risks by two different methods: by sources and media, and by pollutant. In the sources and media ranking, the “residential and consumer product releases to air (indoor air)” category was ranked in the high risk group, along with some outdoor air pollution categories. In the pollutant rankings, ETS, radon, PM, and VOCs were all ranked in the high-risk category, and carbon monoxide and lead were ranked in the medium risk category. The results of the CCRP had a somewhat more substantial scientific basis than most other comparative risk projects, because it was based to a greater degree on actual measurement data, and used distributions of exposure and risk, rather than just population averages. Additionally, it was reviewed by an external scientific review committee.

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The U.S. EPA's 1987 national comparative risk project also ranked indoor air pollution high relative to other environmental areas: indoor pollution other than radon was ranked fourth among the top thirteen national environmental problem areas assessed for cancer risk (U.S. EPA, 1987a). Radon was ranked first, but the risk from radon has been subsequently re-examined and reduced. A number of other states and regions have conducted comparative risk projects as well, with results similar to those of the U.S. EPA and California.

The high ranking of indoor pollution relative to other environmental problems is not surprising, because there are numerous sources of pollutants indoors, indoor air concentrations of some pollutants are often high enough to pose a health risk, and people spend most of their time indoors. The total quantity of air pollutants emitted indoors is much less than that emitted by outdoor sources. However, once emitted, indoor pollutants are diluted much more slowly than pollutants from outdoor sources. When this factor is combined with the fact that Californians, like others from industrialized nations, spend most of their time indoors, there is a much higher likelihood that people will be exposed to pollutants emitted indoors than those emitted outdoors.

Investigators have developed concepts and terms to quantify the portion of pollutant emissions actually inhaled (Bennet *et al.*, 2002; Lai *et al.*, 2000). The inhalation transfer factor (ITF) is defined as the pollutant mass inhaled by an exposed population per unit mass emitted from an air pollution source (Lai *et al.*, 2000).

### *The Rule of 1000:*

***A typical pollutant release indoors is 1000 times as effective in causing human exposure as the same release to urban air.***

KR Smith, in Air Pollution: Assessing Total Exposure in the United States, *Environment*, 30 (8): 10, 1988.

Although more people are exposed to a pollutant released outdoors, the concentration is usually reduced due to wide dispersion, relative to a pollutant emitted indoors. Calculated inhalation transfer factors were several orders of magnitude greater for pollutants emitted indoors and in vehicles than those emitted outdoors, thus indicating a significantly larger fraction of pollutant is inhaled when it is released indoors as opposed to outdoors.

Investigators calculate that pollutants emitted indoors have a 1000-fold greater chance of being inhaled than do those emitted outdoors (Smith, 1988; Bennet *et al.*, 2002; Lai *et al.*, 2000). Thus, reducing indoor emissions by a given amount might be anticipated to have a greater impact on reducing exposure than would reducing outdoor emissions by that same amount. Regulation of outdoor sources such as motor vehicles and industrial plants has notably reduced many outdoor pollutant levels in California. Now, there are significant gains to be achieved in public health protection from reductions in indoor source emissions and other measures that might be taken to reduce indoor concentrations and exposures.

The health effects of indoor pollutants range from irritant effects to respiratory disease, cancer, and even sudden death. Indoor sources of pollutants are numerous, such as building materials, consumer products of all types, combustion appliances, and even some so-called "air fresheners". Common indoor activities such as cooking, cigarette smoking, burning candles, and vacuuming also generate pollutants. The health effects of indoor pollutants, and indoor pollutant sources and concentrations in California, are discussed in Chapter 2 of this report.

## 1.2 PEOPLE SPEND MOST OF THEIR TIME INDOORS

A key reason indoor pollution is so critical to health is that Californians, like others from industrialized nations, spend most of their time indoors – about 87%, on average. So, if pollutants are present indoors, there is a high likelihood that people will be exposed to them. As shown in Table 1.1, California adults spend an average of about 62% of their time in their home. Children spend even more time in their home; infants up to 2 years of age spend 85% of their time inside the home, on average. Thus, the home is a critical exposure microenvironment for all, and especially for children.

**Table 1.1: Average Percent of Time Californians Spend in Major Locations**

AGE	AVERAGE PERCENT OF TIME			
	Inside the Home	Other Indoors	Outdoors	Inside a Vehicle
<b>Children<sup>1</sup></b>				
<b>0 - 2</b>	<b>85</b>	<b>4</b>	<b>7</b>	<b>4</b>
<b>3 - 5</b>	<b>76</b>	<b>9</b>	<b>10</b>	<b>5</b>
<b>6 - 11</b>	<b>71</b>	<b>12</b>	<b>13</b>	<b>4</b>
<b>All Children (0 - 11)<sup>1</sup></b>	<b>76</b>	<b>10</b>	<b>10</b>	<b>4</b>
<b>Adults and Teens<sup>2</sup></b>	<b>62</b>	<b>25</b>	<b>6</b>	<b>7</b>

1. From: *Study of Children's Activity Patterns* (Wiley *et al.*, 1991a, ARB Contract No. A733-149; Phillips *et al.*, 1991).
2. From: *Activity Patterns of California Residents* (Wiley *et al.*, 1991b, ARB Contract No. A6-177-33; Jenkins *et al.*, 1992a).

## 1.3 CHILDREN'S HEALTH – VULNERABILITY IMPLIES INCREASED INDOOR AIR QUALITY IMPACT

In recent years there has been an increasing awareness that children may be more susceptible than adults to the harmful effects of air pollution. Additionally, children are more highly exposed to some indoor contaminants than are adults. Thus, children are likely at greater risk from indoor pollution than adults.

- **Children's physiology and developing lungs and bodies make them more susceptible to chemicals that affect development and lung function.** Children's immune systems are not fully developed and their growing organs and structures are more easily harmed. For example, lead is more readily absorbed from the digestive tract of children, and the developing central nervous system is more susceptible to damage than that of an adult. Pollutants that cause irritation or inflammation in the airways are more likely to obstruct a child's airways because they are narrower than airways of an adult. Results from the ARB-funded Children's Health Study indicate the lungs of children in high-pollution communities develop more slowly and move air less efficiently than lungs of children in low-pollution communities (Gauderman *et al.*, 2000). When exposed to ETS, children are at greater risk than adults for developing lower respiratory tract infections, bronchitis, pneumonia, fluid in

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the middle ear, and asthma symptoms (NCI, 1999). Additionally, young children appear to be more susceptible to the effects of environmental tobacco smoke (ETS) than older children (IOM, 2000).

- **Infants and children inhale more air relative to their size** than do adults at a given level of activity (ARB/OEHHA, 2000; Adams, 1993). Additionally, children often breathe through their mouths, bypassing the filtering effect of the nose and allowing more pollutants to be inhaled. Thus, for the same amount of time spent in a given location/activity as an adult, a child will inhale a greater quantity and thus receive a greater dose of the chemicals in the air. Children also have a larger lung surface area per unit of body weight, relative to adults. This contributes to a higher breathing rate/unit surface area and elevated exposure in children (Thurlbeck, 1988; Plopper and Thurlbeck, 1994).
- **Children's activities bring them into close proximity to indoor sources.** First, infants and young children spend more time indoors at home (see Table 1.1) than do adults. Additionally, younger children spend more time near indoor sources such as operating gas stoves, e.g., near the parent while cooking (Phillips *et al.*, 1991), leading to higher exposures to nitrogen dioxide and other cooking emissions.
- **Also, children spend more time on floors and more frequently put fingers and objects into their mouth** (Zartarian *et al.*, 1998; Zartarian and Leckie, 1998). This can lead to additional dermal and ingestion exposure to airborne TACs deposited and adsorbed onto floor dust, such as lead and other toxic metals, PAHs, and pesticides. Because the breathing zone for an infant or small child is several inches to a foot or so above the floor, while that for adults is several feet above the floor, particles stirred up by activity may become available to be inhaled by the child but are not elevated in the breathing zone of adults (Bearer, 1995).

Concerns about children's health prompted the California Legislature to pass the Children's Environmental Health Protection Act (SB 25, Escutia) in 1999. This Act requires Cal/EPA to specifically consider children when setting Ambient Air Quality Standards (AAQS) and reviewing pollutants for identification or regulation as Toxic Air Contaminants (TACs). As required by this legislation, ARB and OEHHA have re-evaluated the AAQS for particulate matter and are currently evaluating the AAQS for ozone and nitrogen dioxide. OEHHA has identified dioxin, lead, polycyclic organic matter, diesel exhaust particles, and acrolein as the top 5 priority TACs that may impact infants and children the most (OEHHA, 2001). The ARB is beginning to assess the adequacy of existing control measures for these compounds relative to the health of children.

### 1.4 ENVIRONMENTAL JUSTICE CONSIDERATIONS RELATIVE TO INDOOR AIR QUALITY

Senate Bill 115 (1999) directs the California Environmental Protection Agency to design an environmental justice mission statement for boards, departments, and offices within the agency. State law (California Government Code § 65040.12c) defines environmental justice as the fair treatment of people of all races, cultures, and incomes with respect to the development, adoption, implementation, and enforcement of environmental laws, regulations, and policies. In response to SB 115, the ARB approved *Policies and Actions for Environmental Justice* in December, 2001. This document establishes a framework for incorporating environmental justice into all ARB programs, policies, and regulations (ARB, 2001a).

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The ARB has taken several steps to directly and indirectly address environmental justice concerns related to indoor air quality. Special air-monitoring studies have been conducted in classrooms, homes, and at schools in communities located near industrial sources of pollution and/or heavy vehicular traffic. Preliminary results indicate pollutant levels in the selected communities are similar to levels in other communities. To assure that information is available to all stakeholders, documents designed for public education, such as fact sheets and a school advisory, have been published in both English and Spanish.

Census statistics indicate that the poor are more than three times as likely (22% versus 7%) to have substandard-quality housing (Evans and Kantrowitz, 2002) and that blacks and low-income persons are more likely than the general population to be in housing with severe physical problems (Krieger and Higgins, 2002). Children in low-income families may bear additional burdens because they are more likely to be in school buildings that have environmental problems; poor plumbing, inadequate heating, and poor indoor air quality (Evans and Kantrowitz, 2002).

Research is needed to determine the complex relationship between socioeconomic status (SES), environmental factors, and health status, particularly as they relate to indoor air pollution. To date, only factors related to the prevalence of asthma have received substantial study. The prevalence of asthma appears to be more strongly correlated with lower socioeconomic status than with race and ethnicity (IOM, 1993). Yet, California data show that African Americans, American Indians, and Alaska Natives experience a higher prevalence of lifetime asthma than other groups in the population (Meng *et al.*, 2003). Identifying the contribution of exposure to biological agents to poor health is difficult. The increase in asthma prevalence is greatest in children from lower income homes, probably due to an increase in prevalence of allergic conditions in their homes (Auinger *et al.*, 2003). Dust mites and cockroaches are important triggers for asthmatics that are more likely to be present in urban settings (IOM, 1999). Reviewing data accumulated in the second National Health and Nutrition Examination Survey (NHANES II) and the Harvard Six Cities Study, Eggleston (2000) concluded that ethnicity, poverty, and residence combined to influence asthma prevalence in inner-city children in ways that could not be easily disentangled (IOM, 2004).

“Economic factors [also] may encourage poor building practices. Combinations of pressure to build quickly and cheaply can result in poorly constructed buildings that are more likely to have water leaks.... Poverty combined with the lack of affordable housing may also create incentives to forgo or limit investment in maintenance that might help to prevent moisture problems and subsequent adverse impacts on the health of the occupants” (IOM, 2004). Those who live in substandard housing are exposed to more pests, as well as pesticides to get rid of them (Flynn *et al.*, 2000). Pesticides adsorb onto particles and accumulate in the carpet, where children and others can be exposed to them. In an effort to improve their environment, a higher percent of low-income individuals use room fresheners – products that may introduce additional toxic chemicals to the indoor environment (Wiley *et al.*, 1991b).

Children living in urban areas are disproportionately exposed to lead, primarily from lead-containing paint that has been used on older houses. Exposure to lead in both house dust and air takes a toll on children. Research indicates blood lead levels are higher for poor and minority children in central cities (IOM, 1999).

## 2. HEALTH EFFECTS, SOURCES AND CONCENTRATIONS OF INDOOR AIR POLLUTANTS

Emissions from indoor sources contribute to exposure and risk in two ways. Some pollutants are emitted in substantial quantities over extended periods from large surfaces, such as formaldehyde from composite wood products made with urea-formaldehyde resin. These sources contribute to elevated indoor pollutant levels in many buildings where a large portion of the population spends their time. Large numbers of individuals can be affected due to the large quantity of indoor emissions. Other products, such as aerosol sprays or solvents, emit much smaller quantities of pollutants, and are used by a subset of the population. However, those who use such products use them in such a manner that the chemicals emitted are released near the user's breathing zone (area near the nose and mouth). A high concentration of the chemical consequently may be inhaled during product use before the chemical has a chance to dilute in the air.

It is important to note that health effects are determined not only by the specific toxicology of the air pollutant, but also by the exposure and absorbed dose. The higher the exposure and dose, the higher the risk of adverse health effects. In addition, more severe effects generally occur with higher doses. It is not possible in this document to describe the dose-response relationship for all indoor air pollutants. Further information on dose-response relationships can be found on the OEHHA website ([www.oehha.ca.gov](http://www.oehha.ca.gov)).

Hundreds of substances representing a range of chemical, physical, and biological species have been identified as indoor air pollutants. Indoor air pollutants include volatile organic compounds (VOCs), inorganic gases, particulate matter (PM), and complex mixtures such as environmental tobacco smoke (ETS). Indoor concentrations of many pollutants are often higher than outdoor concentrations due to the use of indoor pollutant sources in a confined space. Some pollutants such as formaldehyde, radon, asbestos, cigarette smoke, and mold have received a substantial amount of study. Other pollutants have received minimal study, and undoubtedly some pollutants have not yet been identified.

Indoor air pollution can cause a variety of adverse impacts on human health, from irritant effects to respiratory disease, cancer, and death. The major health effects that can occur from exposure to common indoor pollutants are indicated in Table 2.1. These effects have a major impact on Californians' health each year, and most can be readily avoided. Asthma, cancer, irritant effects, and sick building syndrome are discussed below. Later in this chapter the more common indoor pollutants, their sources, and indoor concentrations are discussed.

### 2.1 KEY HEALTH IMPACTS

#### 2.1.1 Asthma

Asthma is a chronic inflammatory lung disease that results in partially reversible constriction of the airways. It is characterized by episodes of wheezing, shortness of breath, or coughing that may occur at any time. Asthma is a critical health issue because of its negative impact on the quality of life, increased morbidity and mortality, and substantial economic impact.

**Table 2.1. Sources and Potential Health Effects of Major Indoor Air Pollutants**

<b>POLLUTANT</b>	<b>MAJOR INDOOR SOURCES</b>	<b>POTENTIAL HEALTH EFFECTS ASSOCIATED WITH ONE OR MORE OF THE POLLUTANTS LISTED*</b>
<b>Asbestos</b>	Building materials in older homes released during renovation, naturally occurring in some soils	Lung cancer, asbestosis, mesothelioma
<b>Biological Agents</b> (bacteria, fungi, viruses, house dust mites, animal dander, cockroaches, microbial VOCs)	House and floor dust, pets, bedding, poorly maintained air- conditioners, humidifiers, dehumidifiers, moist structures or furnishings, insect infestation, building occupants	Allergic reactions; asthma, eye, nose, and throat irritation, humidifier fever, influenza, and other infectious diseases
<b>Carbon Monoxide</b>	Unvented or malfunctioning gas and propane appliances, wood stoves, fireplaces, tobacco smoke, motor vehicles in attached garages	Headache, nausea, angina, impaired vision and mental functioning, fatal at high concentrations
<b>Endocrine Disruptors</b> (phthalates; DDT, chlordane, heptachlor, o-phenylphenol, PBDEs)	Plastics, pesticides, flame retardants	Mimic or block natural effects of hormones (estrogen and others); developmental abnormalities
<b>Environmental Tobacco Smoke (ETS)</b>	Cigarettes, cigars, and pipes	Respiratory irritation, bronchitis and pneumonia in children, asthma in preschool children, lung cancer; heart disease, aggravated asthma, decreased lung function
<b>Formaldehyde, Other Aldehydes</b>	Composite wood products such as plywood and particleboard, furnishings, wallpaper, durable press fabrics, paints, combustion appliances, tobacco smoke	Cancer, eye, nose, and throat irritation, headache, allergic reactions, aggravated asthma, decreased lung function
<b>Lead</b>	Lead paint chips, contaminated soil	Learning impairment
<b>Nitrogen Dioxide</b>	Unvented or malfunctioning gas appliances, other combustion appliances	Aggravated asthma, decreased lung function, eye, nose, and throat irritation, increased respiratory disease in children
<b>Organic Chemicals</b> (benzene, chloroform, para-dichlorobenzene, methylene chloride, perchloroethylene, phthalates, styrene, others)	Solvents; glues, cleaning agents, pesticides, building materials, paints, treated water, moth repellents; dry-cleaned clothing, air fresheners	Cancer; eye, nose, throat irritation, aggravated asthma, decreased lung function; headaches, at high levels: loss of coordination, damage to liver, kidney and brain
<b>Ozone</b>	Infiltration of outdoor air, ozone generating air “purifiers”, office machines	Lung inflammation, aggravated asthma, cough, wheeze, chest pain
<b>Particulate Matter</b>	Cigarettes, wood stoves, fireplaces, cooking, candles, aerosol sprays, house dust	Increased mortality and hospital admissions, lung cancer, eye, nose, throat irritation, increased susceptibility to sinus and respiratory infections, bronchitis; aggravated asthma, decreased lung function
<b>Polycyclic Aromatic Hydrocarbons (PAH)</b>	Cigarette smoke, cooking, woodburning	Cancer, gene mutation
<b>Radon</b>	Uranium-bearing soil under buildings, ground-water, construction materials	Lung cancer (especially in smokers)

\* Please note that when multiple pollutants are listed in a group, each pollutant may not cause all of the health effects listed in the third column.

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A number of indoor pollutants can cause or exacerbate asthma and chronic bronchitis. Indoor biological agents are associated with these diseases (IOM, 2000); however, it is clear that biological agents alone cannot explain the increase in asthma over the last few decades. The recent rise in asthma prevalence has been too rapid to be attributed to genetic factors and biological allergens alone: indoor and outdoor air pollution have been identified as potentially important contributors to the increase of asthma (McConnell *et al.*, 2002; Platts-Mills and Carter, 1997; Duhme *et al.*, 1998; Karol, 2002).

Over the past three decades, asthma prevalence has been on the rise in industrial nations, and the death rate due to this disease has doubled (Karol, 2002). The Centers for Disease Control and Prevention (CDC) reported that in the year 2000, asthma was responsible for 4,487 deaths in the U.S., as well as approximately 465,000 hospitalizations, 1.8 million emergency department visits, and 10.4 million visits to physicians across all age groups (CDC, 2003). Currently, about 7.2% of adults in the U.S. have asthma (CDC, 2003). According to 2001 data, 11.9% of Californians, or 3.9 million people, have asthma (CHIS, 2003). California also has higher rates of asthma mortality than the nation as a whole. The reasons for these elevated rates in California are unknown at this time. Children have been particularly hard-hit; from 1980 to 1994, there was a 160% increase in asthma prevalence in those up to 4 years of age in the U.S. (Mannino *et al.*, 1998). In California, asthma prevalence is highest among children 12-17 years of age. Asthma is one of the leading causes of school absenteeism in children, and results in missed workdays and lost productivity in adults. Asthma affects all races and ethnic groups, and both genders. Every year about 40,000 Californians are hospitalized and about 500 Californians die because of asthma. Asthma hospitalization and death rates are higher among African-Americans compared to other racial groups. Further, hospitalization rates among children are much higher than other age groups. Although the causes for the observed increase in asthma prevalence, hospitalizations, and death are not fully identified, indoor air pollution has been identified as a contributing factor.

In a recent report by the National Academy Institute of Medicine, entitled *Clearing the Air: Asthma and Indoor Air Exposures* (IOM, 2000), the Committee on the Assessment of Asthma and Indoor Air examined the scientific literature relating indoor air pollutants and other factors to asthma. Their key findings are shown in Tables 2.2 and 2.3. Recent reviews of the medical literature have also established that building dampness approximately doubles the risk for respiratory symptoms in building occupants and can exacerbate asthma. The agent(s) responsible for dampness-related increased risk for asthma exacerbation have not been conclusively identified but may involve some or all of the following: house dust mites, microbiological agents such as fungi or bacteria, or organic chemicals released during degradation of building materials or furnishings (Bornehag, 2001; 2004).

The IOM committee found that, in addition to the known biological asthma triggers such as house dust mites, cockroaches, and animal dander, chemicals in ETS can exacerbate asthma in preschool children. Evidence for an association between formaldehyde exposure and wheezing is limited, often due to confounding factors such as exposure to multiple pollutants. Airway responses such as wheezing, waking with shortness of breath, and asthma attacks have been associated with gas stove use (Jarvis *et al.*, 1996). Sufficient evidence exists to conclude that ETS can exacerbate asthma in preschool-aged children and provides an association between ETS exposure and the development of asthma (IOM, 2000). However, scientists found only limited or suggestive evidence of ETS as an asthma trigger in older children and adults, and insufficient evidence to consider it a causal factor in these groups (IOM, 2000). The IOM committee noted that studies have linked outdoor PM with respiratory problems, and that outdoor fine particles, PM<sub>2.5</sub>, readily enter the indoor environment. Studies that have addressed



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asthma specifically have found some evidence for asthma exacerbation due to outdoor PM exposure (IOM, 2000).

Although the Committee on the Assessment of Asthma and Indoor Air found inadequate evidence to link VOCs with exacerbation of asthma, more recent studies have found an association between residential VOCs to asthma and its symptoms. Delfino (2002) published a review of the epidemiological evidence for links between air toxics and asthma. Delfino cites Swedish studies that showed that self-reported asthma prevalence in school children increased with increasing VOC levels, and asthmatic adult symptoms occurred in association with toluene, C<sub>8</sub>-aromatics, terpenes, formaldehyde, and limonene. Adult asthma prevalence, wheeze, and blood eosinophil concentrations were higher in newly painted homes, consistent with higher VOC levels (particularly 2,2,4-trimethyl 1,3-pentanediol diisobutyrate and formaldehyde) measured in such homes. In another European study cited by Delfino, elevated levels of benzene and styrene were associated with respiratory infections in newborns with compromised health. The newborns had either low birth weight or an abnormal immune response (indicated by high levels of IgE in cord blood). In the same study, wheezing was related to house painting and carpet installation during the first year of life. Delfino cautions that in these studies, the effects seen may be subject to confounding by other causal agents.

**Table 2.2. Indoor Exposures and Exacerbation of Asthma**

<b>Sufficient Evidence of a Causal Relationship</b> <ul style="list-style-type: none"><li>• Cat</li><li>• Cockroach, House dust mite</li><li>• ETS (preschoolers)</li></ul>
<b>Sufficient Evidence of an Association</b> <ul style="list-style-type: none"><li>• Dog</li><li>• Fungi or molds, Rhinovirus</li><li>• NO<sub>2</sub>, NO<sub>x</sub> (high-level exposures)</li></ul>
<b>Limited or Suggestive Evidence of an Association</b> <ul style="list-style-type: none"><li>• Domestic birds</li><li>• <i>Chlamydia pneumoniae</i>, <i>Mycoplasma pneumoniae</i>, Respiratory Syncytial Virus (RSV)</li><li>• ETS (school-aged children and adults)</li><li>• Formaldehyde, Fragrances</li></ul>
<b>Inadequate or Insufficient Evidence to Determine Whether or Not an Association Exists</b> <ul style="list-style-type: none"><li>• Cow, Horse, Rodents</li><li>• <i>Chlamydia trachomatis</i>, Endotoxins</li><li>• Houseplants, Pollen</li><li>• Pesticides, Plasticizers, VOCs</li><li>• Insects other than cockroaches</li></ul>

Source: IOM, 2000

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Delfino's (2002) review identified several links between asthma symptoms and indoor air pollutants, especially formaldehyde:

- A relationship exists between formaldehyde exposure and occupational asthma.
- Children in homes with formaldehyde concentrations greater than 41 ppb are more often diagnosed with asthma and chronic bronchitis.
- Adults show more wheeze, chronic cough, and lower peak expiratory flow in homes with higher formaldehyde concentrations.
- Non-asthmatics in homes with formaldehyde levels of 50 ppb or higher have elevated levels of expired nitric oxide, a marker for lower airway inflammation.

**Table 2.3. Indoor Exposures and Development of Asthma**

<b>Sufficient Evidence of a Causal Relationship</b> <ul style="list-style-type: none"><li>• House dust mite</li></ul>
<b>Sufficient Evidence of an Association</b> <ul style="list-style-type: none"><li>• ETS (preschoolers)</li></ul>
<b>Limited or Suggestive Evidence of an Association</b> <ul style="list-style-type: none"><li>• Cockroach (preschoolers)</li><li>• Respiratory Syncytial Virus (RSV)</li></ul>
<b>Inadequate or Insufficient Evidence to Determine Whether or Not an Association Exists</b> <ul style="list-style-type: none"><li>• Cat, Cow, Horse, Dog, Domestic birds, Rodents</li><li>• Cockroaches (except for preschoolers)</li><li>• Fungi or mold, <i>Chlamydia pneumoniae</i>, <i>Chlamydia trachomatis</i>, <i>Mycoplasma pneumoniae</i>, Endotoxins</li><li>• Houseplants, Pollen</li><li>• NO<sub>2</sub>, NO<sub>x</sub></li><li>• Pesticides, Plasticizers, VOCs, Formaldehyde, Fragrances</li><li>• ETS (school-age and older)</li></ul>

Source: IOM, 2000

The association between VOCs and asthma is complex. Other reviews support the association between VOCs and symptoms of asthma (Duhme *et al.*, 1998; Leikauf, 2002). Delfino *et al.* (2003b) studied Hispanic children with mild asthma in a Los Angeles community with high VOC levels near major freeways. Bothersome or more severe asthma symptoms were associated with breath concentrations of benzene, but not other breath VOCs. On breath sample days, asthma symptoms were also associated with 1-hour ambient NO<sub>2</sub> and sulfur dioxide (SO<sub>2</sub>).

Studies conducted in the workplace also demonstrate an association between asthma symptoms and chemicals used indoors. Between 1993 and 1997, 12% of confirmed cases (236 of 1,915) of work-related asthma in California, Massachusetts, Michigan, and New Jersey were related to exposure to cleaning products. Chlorine bleach was identified as the cleaning agent associated with the greatest number of cases. Exposures were greatest in medical settings, schools, and hotels (all non-industrial workplaces) with janitors, cleaners, and housekeepers experiencing the highest incidence of the disease (both new-onset and work-aggravated cases

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of asthma: 80% were new onset; Rosenman *et al.*, 2003). From 1993 through mid-2003, 3,188 cases of work-related asthma were identified from doctor's first report of occupational injury or illness (DFR) in California (DHS, 2004). These cases are not specific to cleaning products.

### 2.1.2 Cancer

Many indoor air pollutants are known or suspected carcinogens. Formaldehyde, benzo(a)pyrene and other polycyclic aromatic hydrocarbons (PAHs), environmental tobacco smoke, benzene, chlorinated solvents such as tetrachloroethylene, and radon are a few of the identified carcinogens commonly found in indoor air, some at levels much higher than outdoor levels. Several technical documents provide summary data regarding the carcinogenic potential of these pollutants. Cancer unit risks and potency factors for 121 of the 201 carcinogenic substances for which emissions must be quantified in the California Air Toxics Hot Spots program are provided in the *Technical Support Document for Describing Available Cancer Potency Factors* (OEHHA, 2002; <http://www.oehha.ca.gov/air.html>). The U.S. EPA's Integrated Risk Information System (IRIS) provides a similar list of cancer potency values (<http://www.epa.gov/iris/>).

Risk assessments are conducted to estimate the increased risk of health problems in people who are exposed to different amounts of toxic substances. Risk is dependent on the amount of a pollutant people actually inhale, which depends on the air concentration of the pollutant in a given environment, the length of time a person is in that environment, and the person's breathing rate during that time. Since people spend the majority of their time indoors, moderate and high concentrations of indoor pollutants generally translate to elevated risk.

Several field studies have measured indoor concentrations of carcinogenic chemicals in California (Wallace *et al.*, 1988; Wallace *et al.*, 1991a; Sheldon *et al.*, 1992a; Avol *et al.*, 1996; and others). Results of these studies indicate that carcinogens are routinely found in most homes, often at levels higher than outdoor levels, due to the presence of indoor sources. Table 2.4 lists key pollutants identified in California studies, and indicates their cancer classification by the International Agency for Research on Cancer (IARC), a part of the World Health Organization. For the most recent classifications, visit the IARC website. Table 2.4 also lists the status of each pollutant as a California toxic air contaminant (TAC).

ARB staff estimate that about 230 excess cancer cases occur per year in California from indoor sources of toxic air contaminants, not including the excess cancer from exposure to radon, environmental tobacco smoke (ETS), and some other indoor carcinogens. This estimate of 230 cancer cases per year is based on risk estimates from the 1994 California Comparative Risk Project (CCRP, 1994), updated to reflect reduced exposure and risk from indoor formaldehyde. Formaldehyde levels are estimated to have decreased by about one-half since the 1980s studies on which the 1994 estimate was based (see Appendices II and III). The chemicals with the highest estimated risk in the CCRP were formaldehyde (found in many building materials and consumer products) and para-dichlorobenzene (used in mothballs and air fresheners). Other chemicals included were perchloroethylene (used in dry-cleaning), chloroform (a by-product of water chlorination and use of chlorine in spas and washing machines), trichloroethylene, benzene, 1,3-butadiene, styrene, benzo(a)pyrene, and di-2-ethylhexylphthalate (DEHP—a plasticizer). The latter organic chemicals are found in many different consumer products and building materials, and some also are produced by combustion processes (such as when cooking food or burning wood). (DEHP is listed as a California TAC. However, the International Agency for Research on Cancer (IARC) recently determined that there is inadequate evidence for the carcinogenicity of DEHP in humans, yet sufficient evidence

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in experimental animals for carcinogenicity (<http://www-cie.iarc.fr/htdocs/monographs/vol77/77-01.html>).

The 1994 CCRP estimates, like other cancer risk estimates, were derived using 95% upper-bound cancer potency factors, combined with measured indoor exposure distributions. Cancer risk methodology based on upper-bound cancer potency estimates provides a common, protective basis for comparing risks across topic areas. However, the risk estimates should not be interpreted as predictions of actual disease (CCRP, 1994). The risk can be much lower, depending on the actual dose of the pollutant inhaled and absorbed, and other factors.

**Table 2.4. Carcinogenic Status of Selected Indoor Air Pollutants**

Compound	Cancer Status <sup>1</sup> Classification of International Agency for Research on Cancer (IARC) <sup>2</sup>	Status as a California Toxic Air Contaminant
Acetaldehyde	Group 2B, possible human carcinogen	Yes
Asbestos	Group 1, known human carcinogen	Yes
Benzene	Group 1, known human carcinogen	Yes
Benzo(a)pyrene (PAHs)	Group 2A, probable human carcinogen	Yes
Chloroform	Group 2B, possible human carcinogen	Yes
<i>p</i> -Dichlorobenzene	Group 2B, possible human carcinogen	Yes
Di-2-ethylhexylphthalate	Group 3, not classified	Yes
Environmental Tobacco Smoke	Group 1, known human carcinogen	Under consideration
Formaldehyde	Group 1, known human carcinogen	Yes
Methylene chloride	Group 2B, possible human carcinogen	Yes
Perchloroethylene	Group 2A, probable human carcinogen	Yes
Radon	Group 1, known human carcinogen	Yes
Styrene	Group 2B, possible human carcinogen	Yes
Trichloroethylene	Group 2A, probable human carcinogen	Yes

<sup>1</sup>Source: OEHHA, 2002; IARC website

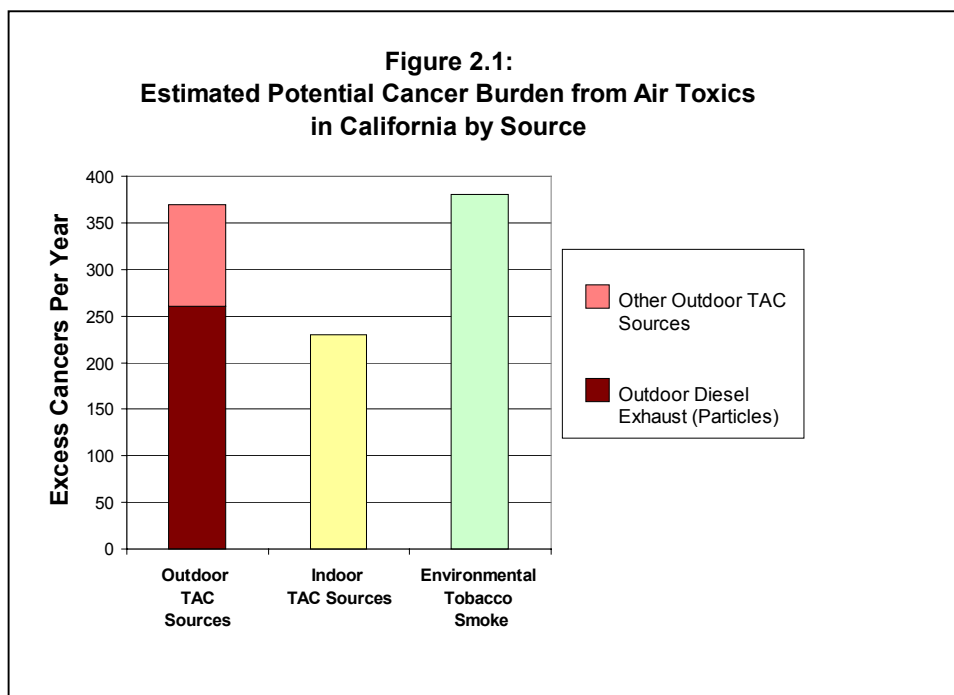
<sup>2</sup>Group 2A compounds have limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. Group 2B compounds have limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals.

Since the time the studies used for the CCRP were conducted, some levels of indoor pollutants are estimated to have decreased while others likely have increased. However, there are insufficient new data available to refine the 1994 estimates for those chemicals. Because changes likely have occurred in both directions and are likely to be relatively small, and because the 1994 CCRP estimates did not include all known indoor carcinogenic pollutants (methylene chloride, other polycyclic aromatic hydrocarbons, and other phthalates were not included, for example), the 1994 estimates remain the best available estimates for the overall cancer risk posed by indoor chemical pollution in California, excluding that from radon gas, environmental

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tobacco smoke, and asbestos (discussed later in this document). The details of ARB's assessment using the CCRP results are provided in Appendix II.

This estimate of 230 excess cancer cases approaches the cancer burden from diesel exhaust particles, which is estimated to result in 260 excess cases per year in California, and exceeds the cancer risk for other outdoor pollutants, estimated at about 110 cancer cases per year (Figure 2.1). Indoor air cancer risk is also nearly two-thirds of the total cancer risk estimated for outdoor pollutants. It is estimated that cancer risk due to diesel exhaust particles will decrease 75% by 2010 (ARB, 2000a), leaving indoor air as a predominant source of air pollution cancer risk.



Exposure to environmental tobacco smoke (ETS) makes a significant contribution to the cancer burden from air pollution as well. Although smoking prevalence and exposure of non-smokers has decreased in California, preliminary updated exposure and risk estimates for ETS developed by OEHHA are similar to those estimated in 1997 (OEHHA, 1997), due to the increase in the California population (and thus the number of individuals exposed). Updated estimates, which are currently undergoing peer review, show 380 excess lung cancer cases per year (OEHHA, 2004). This ETS risk level is similar to the total outdoor air pollutant cancer burden; however, because workplace exposure has decreased to nearly zero since the mid-1990s, and the prevalence of smoking has decreased substantially as well, the current cancer burden from ETS may be somewhat lower than shown in this graphic. Nonetheless, the contribution of ETS will remain significant for some time, because some individuals (including children) are still exposed to substantial levels of ETS.

Payne-Sturges *et al.* (2004) recently calculated cancer risk associated with indoor and personal exposure levels of VOCs and found risk levels similar to those estimated in this report. They measured the personal, indoor, and outdoor concentrations of 11 VOCs for 33 non-smoking adults in South Baltimore, Maryland. VOC concentrations were similar to earlier reported measurements in the California VOC Total Exposure Assessment Methodology (TEAM) studies,

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with some variations noted both higher and lower. For personal monitoring, the highest median cancer risks were attributed to chloroform, benzene, and carbon tetrachloride. The authors assumed the cancer risks for the 11 VOCs were additive and calculated 165 lifetime cumulative cancer risks per 1 million based on average indoor concentrations of 11 measured VOCs, with formaldehyde not included. Calculations based on personal exposures and outdoor concentrations were 183 and 43 cumulative lifetime cancer risks per 1 million, respectively. A background risk (or ambient risk) was not subtracted from the indoor calculation. Applying this methodology to California, the estimate is comparable to the 230 excess cancer cases estimated in this report, based on a population of 35 million and the inclusion of formaldehyde, some other VOCs, and semi-volatiles such as benzo(a)pyrene, in the California estimate.

Other recently developed risk estimates also demonstrate the carcinogenic risk posed by indoor pollutants present at average concentrations. In the absence of indoor standards, Hoddinott and Lee (2000) applied U.S. EPA Superfund risk assessment methodology to selected VOCs to determine if indoor concentrations produce significant risks. Indoor VOC concentrations from two studies completed in the 1980s (Wallace, 1987; Cohen *et al.*, 1989) were used to determine the level of risk associated with VOCs measured inside residences. U.S. EPA considers acceptable levels of cancer risk to be one increase in lifetime cancer incidence per 10,000 to 1,000,000 persons (U.S. EPA, 1989). Hoddinott and Lee (2000) calculated that the risk for adults and children, based on average levels found in homes, exceeded the acceptable risk level of one in a million for a number of pollutants. Those pollutants are found in dry-cleaned clothing, ETS, cleaning agents, glued carpet, gasoline, and degreasers. The authors concluded that “Chemical concentrations resulting from ‘off-gassing’ from normal household activities and materials can result in a health risk estimate that exceeds the benchmark used at hazardous waste sites”. The authors also note that “the data used in this evaluation predate changes in the manufacturing of indoor products. These efforts may have reduced some of the emissions in the average home.”

### 2.1.3 Irritant Effects

Many indoor pollutants cause eye, nose, and throat irritation (Wolkoff and Nielsen, 2001). Tables of irritant thresholds have been developed to provide guidance for non-irritating levels of pollutants at workplaces and in the home (Molhave, 1991a; Devos *et al.*, 1990). The OEHHA Chronic Reference Exposure Level for formaldehyde is set at 2.4 ppb to protect against irritation of the eyes and upper and lower respiratory tract. Ozone, the primary ingredient in smog and a strong oxidant, also irritates the respiratory system, causing coughing, throat irritation, or a burning sensation in the airways. Ozone irritation can lead to a feeling of chest tightness, wheezing, and shortness of breath (ARB, 2000b). Other chemicals such as isoprene and terpenes, have been shown to react with oxidants, producing irritating products such as formaldehyde, terpene oxides, and fine particles (Long *et al.*, 2000; Wilkins *et al.*, 2001). Some biological contaminants such as some types of mold are also known to cause irritant effects.

#### 2.1.3.1 Reaction Products

Indoor organic chemicals react with oxidants such as ozone, hydroxyl radicals, and nitrate radicals to produce secondary pollutants. Weschler (2004) provides an excellent review of studies investigating indoor chemical reactions published since 2000. The review focuses on the importance of hydroxyl radicals in indoor reactions, reactions occurring on indoor surfaces, and the impact secondary reaction products have on building occupants. Traditional analytical methods often do not detect some of the short-lived, highly reactive compounds that are produced. Sensory measurements have been used to detect changes in indoor air quality

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associated with the reactions. It is thought that the ozone/terpene reactions dominate indoor chemistry based on the frequent presence of ozone and ubiquitous presence of terpenes in indoor environments (Weschler, 2004).

Terpenes are reactive chemicals (e.g.,  $\alpha$ -pinene, *d*-limonene, myrcene) that are frequently used in cleaning products and other products for their favorable odor characteristics and solvent properties. In order to use less toxic ingredients in consumer products, manufacturers have replaced petroleum-based hydrocarbons with plant-derived compounds, such as *d*-limonene. These compounds are Generally Recognized as Safe (GRAS) by the Food and Drug Administration (FDA). They can be airway irritants at concentrations greater than normally encountered in indoor air (Wolkoff *et al.*, 2000). However, terpenes also have been associated with irritation at lower levels: the irritant chemicals are hypothesized to be a product of the reaction of terpenes with oxidants, rather than the terpene itself (Wilkins *et al.*, 2001; Weschler and Shields, 1997; Weschler and Shields, 1996; Atkinson and Arey, 2003).

Investigators are exploring potential reactions between unsaturated hydrocarbons and oxidants in an effort to more positively identify the cause of sick building health effects. Several reactions, such as between ozone and unsaturated hydrocarbons, ozone and nitrogen oxides, and free radical reactions, may lead to the formation of more irritating indoor compounds. These reactions can lead to the production of submicron particulate matter, aldehydes (formaldehyde) and ketones with lower odor thresholds and greater irritancy than precursors, carboxylic acids such as formic acid and acetic acid, and free radicals (Weschler and Shields, 1997; Sarwar *et al.*, 2004). Pollutants with reactive double bonds such as terpenes and alkenes react with ozone and nitrogen oxides to produce products that result in airway irritation similar to that of formaldehyde. Fan *et al.* (2003) confirmed the reaction of ozone with *d*-limonene and ozone with  $\alpha$ -pinene under indoor conditions to generate submicron particles and other potentially irritating species, such as aldehydes and organic acids. To minimize these reactions, Fan *et al.* (2003) suggest "limiting use of products that emit high-reactivity alkenes during episodes when outdoor ozone levels are elevated", reducing outdoor ozone levels, and minimizing the penetration of ozone from outdoors.

In this growing area of research, investigators have identified an increase in fine particles associated with mopping floors with a pine-scented cleaning product. It is hypothesized that the generation of particles was the result of ambient ozone (up to 48 ppb) reacting with the terpenes in the cleaning product (Long *et al.*, 2000). Sarwar *et al.* (2004) clearly demonstrated the indoor reaction between ozone and terpenes from various consumer products, leading to increases in fine particle mass concentrations. This area of research warrants increased effort in order to understand the association between indoor air pollutants and related health effects.

### 2.1.3.2 Sick Building Syndrome

Sick Building Syndrome (SBS) is a term used to describe a collection of irritant and neurological effects that occur while occupants are in a building, that generally disappear when affected people are out of the building. Specific causes of SBS have not yet been firmly identified. The most common symptoms include eye irritation, congested nose, headache, fatigue, difficulty concentrating, and dry skin (Tenbrinke *et al.*, 1998). SBS differs from building-related illness (BRI; see Biological Contaminants section) in which an identifiable factor causes a specific illness such as bacteria causing Legionnaires' disease or humidifier fever.

In an attempt to identify factors related to SBS, Seppanen and Fisk (2002) reviewed the literature to summarize factors associated with SBS. They found that relative to natural

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ventilation, air conditioning was consistently associated with a statistically significant increase in the prevalence of one or more SBS symptoms, by approximately 30-200%. This finding reinforces the use of ventilation as a mitigation measure for reducing indoor pollution, but still may not identify the primary cause. The review identifies several confounding factors that are not affected by the HVAC type: quantity of carpet or textile surfaces; sealed windows; building age; depth of the building bays; and dusty surfaces. A European review also found an association between ventilation and comfort and health (Wargocki *et al.*, 2002).

Investigators who study indoor reactive chemistry suggest that the degradation products of VOCs may be responsible for the reported SBS symptoms (Carslaw, 2003; Wolkoff *et al.*, 2000; Wolkoff and Nielsen, 2001; Weschler, 2004). These reactions include ozone/terpene reactions with propagation of hydroxyl radicals and reactions on indoor surfaces such as ozone interacting with carpet. The impact that the products of indoor chemistry can have on building occupants has also been studied on a physiological level (Weschler, 2004).

Mendell (1993) conducted a review of the epidemiological literature related to SBS. In reviewing 32 studies, he found consistent findings for an association of SBS symptoms with air-conditioning, carpets, more workers in a space, video display use, and ventilation rates at or below 10 liters/second/person. With specific causes unidentified, Mendell stressed the importance of using prudent design, operation, and maintenance practices to prevent sick building symptoms.

Tenbrinke *et al.* (1998) reported a new approach for using VOC exposure metrics as predictors of SBS. These authors were able to confirm a link between exposure to low level VOCs and SBS symptoms. Apte and Daisey (1999) used the methodology developed by Tenbrinke to identify an association between mucous membrane symptoms and photocopiers. Apte and Daisey also identified a relationship between sore throat symptoms and fresh paint.

Many biological agents can provoke an immunological response, which most frequently takes the form of allergic reactions to the agent. Common symptoms and signs are watery eyes, runny nose, sneezing, nasal congestion, itching, coughing, wheezing, difficulty breathing, headache, and fatigue. Allergic rhinitis (hay fever) and allergic asthma are examples of hypersensitive responses to biological contaminants. Fungal spores, microbial byproducts, dust mites, cat allergen, and pollens are frequently associated with allergic responses.

## 2.2 Traditional (Criteria) Pollutants

Several pollutants for which outdoor air quality standards have been established occur at elevated levels indoors as well, and can pose a serious health risk. Ambient PM has been associated with premature death and serious respiratory and cardiovascular effects in numerous studies. CO can cause near-term death with high exposures of relatively short duration. All of these pollutants can impose serious, non-fatal health impacts: NO<sub>2</sub> from indoor combustion appliances can harm the lungs and other mucous membranes and cause respiratory disease, and ozone can have similar effects at elevated levels. The health effects, indoor sources, and indoor air concentrations of these traditional (criteria) pollutants are discussed below.

### 2.2.1 Particulate Matter

Particulate matter (PM) is broadly defined as any non-gaseous material suspended in the air. PM can include solid material (i.e., dust), liquid material (i.e., a sprayed aerosol), or a



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combination of solid/liquid materials (i.e., a hydrated vehicle exhaust particle). PM is generally classified by its size. PM<sub>2.5</sub> refers to all suspended matter having aerodynamic diameters less than 2.5 microns ( $\mu\text{m}$ : one  $\mu\text{m}$  = one millionth of a meter) and is commonly referred to as 'fine' PM. PM<sub>10</sub> refers to all suspended matter having aerodynamic diameters less than 10  $\mu\text{m}$ . PM between 2.5  $\mu\text{m}$  and 10  $\mu\text{m}$  is commonly referred to as 'coarse' PM. Both federal and state ambient (outdoor) air quality standards incorporate these size distinctions. Recent studies suggest that PM<sub>2.5</sub> mass may be a better indicator than PM<sub>10</sub> mass for predicting potential health effects resulting from ambient (outdoor) PM exposure (Williams *et al.*, 2000abc; Schwartz and Neas, 2000), although some recent studies have specifically linked health impacts to the coarse fraction (Lippmann *et al.*, 2000; Mar *et al.*, 2000; Ostro *et al.*, 2000).

A substantial portion of indoor particles originate outdoors from outdoor sources (Ozkaynak *et al.*, 1996ab; Abt *et al.*, 2000; Long *et al.*, 2000; Liu and Nazaroff, 2001). Outdoor sources of PM that may infiltrate indoors include PM from transportation sources (i.e., gasoline and diesel powered highway vehicles), agricultural activities (i.e., biomass combustion emissions, fugitive dust emissions, pesticide sprays), biogenic emissions (i.e., forest fire smoke), and many others. Further reduction of these concentrations in outdoor air would likewise lower their concentrations in indoor environments. However, there are numerous indoor sources of PM as well. These include combustion devices and activities such as stoves, fireplaces, cigarette smoking, cooking, and candle burning, all of which can produce indoor PM with harmful components similar to those from outdoor air (Lofroth *et al.*, 1991). Indoor particles also include fibrous materials, pollen, mold spores and fragments, and tracked-in soil particles (Wallace, 1996a). These particles become trapped in/on building surfaces, particularly carpets, and have been shown to persist for a very long time, due to the lack of applying effective cleaning and maintenance procedures, and they may be re-suspended into the air. Some can trigger asthma attacks and allergy symptoms, as discussed previously. Others can have a mix of toxic components such as PAHs, lead, and pesticides adsorbed onto them; these components may contribute to serious health effects such as cancer (PAHs) and developmental effects (lead).

Major epidemiologic studies have shown a strong association between ambient (outdoor) PM concentrations and increased death and disease (e.g., Dockery *et al.*, 1993; Pope *et al.*, 1995) and an increase in the rate of death from cardiovascular and respiratory disease (Samet *et al.*, 2000). Indoor PM, particularly from indoor combustion sources, may be similar in composition to outdoor PM, and might be expected to cause the same impacts as outdoor PM. However, research has only recently been undertaken to examine the differences in indoor and outdoor PM composition, and the relative contribution of indoor PM and outdoor PM to the total PM effects of death and disease have not been studied, and are high priorities for further research. Although considerable progress has been made in elucidating the toxicological mechanisms of outdoor PM toxicity, it is difficult to draw inferences between indoor and outdoor PM without focused studies: indoor PM could be more or less toxic than outdoor PM. However, because ambient PM epidemiological studies are based on particle size and include a mix of particles from combustion sources, soil, and other sources, the epidemiological relationships from ambient PM studies and their magnitude should be examined when considering the potential risk from indoor PM. The effects of ambient PM are summarized below, followed by a brief discussion of the potential impacts of indoor PM.

### 2.2.1.1 Death

Both acute and chronic ambient PM exposure have been associated with an increased risk of premature death, primarily in older adults with preexisting heart and/or lung disease. Studies conducted in California, the U.S., and in diverse cities worldwide suggest that risk of death

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increases about 0.25 to 3.5 percent with each  $10 \mu\text{g}/\text{m}^3$  increase in daily mean ambient PM<sub>2.5</sub> concentration (e.g., Burnett and Goldberg, 2003; Dominici *et al.*, 2003; Fairley, 2003; Goldberg and Burnett, 2003; Moolgavkar, 2003; Schwartz, 2003; Ponka *et al.*, 1998). Long-term cohort studies suggest that the increase in risk of death is about 4% with each  $10 \mu\text{g}/\text{m}^3$  increase in annual mean ambient PM<sub>2.5</sub> concentration (Dockery *et al.*, 1993; Krewski *et al.*, 2000; Pope *et al.*, 1995). Meta-analyses of earlier studies suggest that the effects on death are fairly consistent (Ostro, 1993; Dockery and Pope, 1994; Schwartz, 1994), regardless of where the study was performed. About 6500 deaths occur each year in California due to outdoor particulate pollution levels above the State ambient air quality standards (ARB/OEHHA, 2002).

In a recent assessment of global and regional health risks, Cohen *et al.* (2004) used PM<sub>2.5</sub> (measured or estimated) as an index for urban air pollution. They reviewed literature from all parts of the globe, and estimated the portion of death from specified diseases attributable to urban pollution (PM<sub>2.5</sub>). They estimated that pollution in urban areas worldwide causes about 3% of death attributable to cardiopulmonary disease in adults; about 5% of death attributable to cancers of the trachea, bronchus, and lung; and about 1% of death attributable to acute respiratory infections in children. Based on a year 2000 population, this totals about 800,000 excess deaths and 6.4 million disability-adjusted life years. The greatest burden was estimated to occur in the more polluted and rapidly growing cities of developing countries. The authors noted the universality of PM effects found worldwide, despite some differences in ambient PM sources and composition. This lends support to the likelihood of similarities of impacts from ambient and indoor PM. The authors noted that the estimates derived in their analysis are likely an underestimate, and that they cannot be extrapolated to smaller regions or for other purposes.

### 2.2.1.2 Non-lethal Health Impacts

Several hundred studies have been published examining the association between various measures of ambient PM and a variety of adverse health effects other than premature death. The health outcomes associated with ambient PM concentrations include hospitalization and emergency room visits for respiratory or cardiovascular disease; respiratory symptoms, including asthma symptoms; restrictions in activity and school absenteeism; and reduced lung function and other effects in children. Although these effects are not as serious as immediate death, they are serious (some can lead to death) and affect a greater proportion of the population, and thus have a major impact on public health. The key results from some of the published studies include the following:

- **Hospitalization and serious respiratory disease**

- ✓ Studies consistently report associations between both ambient PM<sub>2.5</sub> and PM<sub>10</sub> and hospital admissions for respiratory and cardiovascular illness (e.g., Atkinson *et al.*, 2003; Sheppard *et al.*, 1999; Sheppard, 2003; Ito, 2003; Zanobetti and Schwartz, 2003; Moolgavkar, 2003; Le Tertre *et al.*, 2003). These effects have been reported mainly for people over age 65 who already have some form of cardiopulmonary disease. Respiratory causes of admission include pneumonia, chronic obstructive pulmonary disease (COPD) and asthma, while cardiovascular causes have included general cardiovascular disease, congestive heart failure, stroke, and ischemic heart disease. Overall, ambient PM<sub>10</sub> has been associated with an estimated increase in risk of hospitalization of 1.25% - 5% per  $10 \mu\text{g}/\text{m}^3$  increase in the daily mean ambient PM<sub>10</sub> concentration for respiratory endpoints, and 0.3% to 2.6% for cardiovascular endpoints.

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- ✓ Associations have also been reported between ambient PM<sub>10</sub> and PM<sub>2.5</sub> and emergency department visits, primarily for asthma exacerbation, which may or may not result in hospital admissions (e.g., Lipsett *et al.*, 1997; Delfino *et al.*, 1997).
- **Respiratory symptoms**
  - ✓ Studies have associated ambient PM<sub>10</sub> and PM<sub>2.5</sub> exposure with asthma and respiratory symptoms, for example cough, phlegm, chest pain, or wheeze (e.g., Delfino *et al.*, 2003; Mortimer *et al.*, 2002; Schwartz and Neas, 2000), asthma exacerbation (e.g., Whittemore and Korn, 1980), and use of asthma medications (e.g., Delfino *et al.*, 1996; Pope *et al.*, 1991). People with asthma retain a greater number of ultrafine PM particles than do healthy subjects, thus making them more susceptible to the health effects of air pollution Chalupa *et al.* (2004).
  - ✓ Cellular level effects have also been identified. Exposure to respiratory irritants can result in local airway inflammation, altered epithelial cell permeability, increased mucus secretion, and bronchoconstriction. Disease states such as asthma and chronic bronchitis can adversely affect particle clearance or removal (e.g., Foster, 1999). Also, the viability and functional integrity of cells in the lungs can be adversely affected by ambient PM exposures (e.g., Soukup and Becker, 2001).
- **Work loss, absenteeism, reduced productivity:**
  - ✓ Ostro (1987) and Ostro and Rothschild (1989) reported 10 to 15% reduction in activity due to respiratory-related causes per 10 µg/m<sup>3</sup> of ambient PM<sub>10</sub>.
  - ✓ Ransom and Pope (1992) reported about a 4% increase in absenteeism per 10 µg/m<sup>3</sup> of ambient PM<sub>10</sub> at an elementary school in Utah.
  - ✓ Gilliland *et al.* (2001) reported an increase of 5.7% in total illness-related absences among 4<sup>th</sup> grade school children (ages 9-10) in 12 southern California communities.
- **Effects on children**
  - ✓ Investigators with the ARB-sponsored Children's Health Study found that, among children with asthma, respiratory symptoms increased with increasing ambient particle levels (McConnell *et al.*, 1999). Results also suggest that children who live in communities with high concentrations of ambient PM may have decreased lung function growth compared to children living in communities with lower concentrations of ambient PM (Gauderman *et al.*, 2000; Peters *et al.*, 1999ab). However, both of these results were also true for NO<sub>2</sub> and acid vapor, and the independent effects of the different pollutants cannot be assessed because of high inter-pollutant correlations. Similar results have also been reported by Horak *et al.* (2002) in Austrian children. A recent study by Delfino *et al.* (2004) found clinically relevant decreases in lung function associated with personal PM exposure in schoolchildren with asthma.
  - ✓ Several recent studies have suggested that the unborn may also be at risk of adverse effects from ambient PM pollution, based on statistically significant relationships between outdoor PM concentration and low birth weight (Ritz *et al.*, 2000; Bobak, 2000), premature birth (Bobak, 2000), neonatal death (Penna and

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Duchiade, 1991; Woodruff *et al.*, 1997; Bobak and Leon, 1998), and fetal growth retardation (Dejmek *et al.*, 1999). However, except for Ritz *et al.*, (2000) and Ritz *et al.*, (2002), these studies have been conducted outside the U.S., in areas with higher ambient PM concentrations than those typically observed in the U.S.

The following incidences of illnesses are estimated to occur annually in California due to outdoor PM<sub>10</sub> levels above the State ambient air quality standard.

- 7,900 cases of chronic bronchitis among people age 27 or older,
- 6,000 hospital admissions for cardiopulmonary causes among the elderly (ages 65 or more),
- 1,000 asthma-related hospital admissions among people age 64 or less,
- 2,300 asthma-related emergency room visits among people age 64 or less,
- 340,000 asthma attacks among all ages.

The California ambient air quality standard for PM<sub>2.5</sub> is 12 µg/m<sup>3</sup> (annual mean), and the PM<sub>10</sub> standards are 50 µg/m<sup>3</sup> (24-hour) and 20 µg/m<sup>3</sup> (annual mean). There are no federal or California standards for indoor PM levels. The national ambient air quality standards (NAAQS) for PM<sub>2.5</sub> are 65 µg/m<sup>3</sup> (24-hour average) and 15 µg/m<sup>3</sup> (annual mean), and for PM<sub>10</sub> are 150 µg/m<sup>3</sup> (24-hour on average) and 50 µg/m<sup>3</sup> (annual mean). These levels are often exceeded in California's indoor environments, particularly when indoor sources are used or when particle-generating activities occur.

### 2.2.1.3 Potential Health Impacts of Indoor PM

The serious adverse health impacts of certain indoor PM components are well documented. As discussed in other sections of this document, tobacco smoke particles, radon daughters, metals such as lead, semi-volatiles such as PAHs, and biological components such as pollens and mold all exert serious, sometimes fatal, impacts on human health. However, based on the serious impacts documented for outdoor PM, it is likely that indoor PM is much more than the sum of its parts—there are likely serious effects not yet measured, quantified, or properly accounted for.

As indicated in the preceding section, large numbers of cases of illnesses are expected to occur each year in California due to outdoor particulate matter pollution levels above the State ambient air quality standards (ARB/OEHHA 2002). Indoor PM is comprised of varying proportions of PM of indoor and outdoor origin (discussed below). Because the additional PM burden from indoor sources is generally not well represented in epidemiology studies, indoor PM emissions may be significant contributors to the adverse impacts seen in the epidemiology studies. Additionally, indoor PM may also contribute to premature mortality, hospital admissions, chronic bronchitis, and other effects beyond the levels quantified in the epidemiology studies.

Two groups have recently examined the literature to determine whether there is sufficient information available to permit a rough estimate of the impacts from indoor PM. The first group was a panel of indoor air quality and PM experts convened by ARB in February 2004 to review and assess what is known regarding the impacts of indoor PM on health. The panel found that only one study provides suggestive evidence of the health effects of indoor combustion emissions. In that study of rat alveolar macrophages, investigators found that indoor-generated particles triggered greater production of tumor necrosis factor than did a comparable amount of outdoor PM, suggesting that indoor-generated PM may be more bioactive than ambient particles (Long *et al.*, 2001). This may be due to PM emissions from indoor combustion sources being relatively “fresh”, and small in size. Alternatively, outdoor PM may actually be much more

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toxic than indoor PM, due to emissions from complex sources such as diesel trucks and industrial plants. There is considerable variability in the chemical composition, acidity, and size distribution of outdoor PM depending on geological conditions, traffic mix, meteorological conditions, proximity to major roadways, and significant stationary sources. However, the Cohen *et al.* (2004) study found that PM impacts were essentially global, with few differences across regions, lending support to the likelihood of similar impacts from indoor PM. The panel determined that available studies document the known effects of ETS particles and infectious and allergenic indoor biological contaminants, but that few studies have been designed to specifically identify effects of other types of indoor-generated PM, such as that from candles or woodsmoke. They concluded that research is sorely needed in this area, to determine the actual toxicity of indoor-generated PM, particularly from indoor combustion sources, and the relative toxicities of indoor and ambient PM.

The second group that examined this issue was a European interdisciplinary group of researchers who reviewed the relevant literature to determine whether particle mass, surface area, or number concentration could be used as risk indicators for health effects in non-industrial buildings (Schneider *et al.*, 2003). The group concluded that the study design of most of the reviewed studies was not focused on finding associations between airborne PM and health outcomes, and consequentially, while airborne particles are likely to cause health effects in non-industrial environments, the scientific evidence was inadequate to permit the use of indoor PM mass, surface area, or number concentration as risk indicators for health effects in buildings.

### 2.2.2 Indoor PM Sources and Emissions

Indoor PM concentrations are typically equal to or higher than concurrently measured outdoor levels (see next section), depending on the sources and activities that are present indoors. Outdoor air infiltration and indoor combustion sources such as smoking and cooking are typically the greatest sources of indoor PM (Wallace, 1996a; Ozkaynak *et al.*, 1996ab; Brauer *et al.*, 2000; Abt *et al.*, 2000; Fortmann *et al.*, 2001). Prominent indoor sources include cigarettes, woodstoves, and candles; cooking and cleaning activities (Ozkaynak *et al.*, 1996ab; Abt *et al.*, 2000, 2001; Long *et al.*, 2000); the presence and activities of occupants (Abt *et al.*, 2000; Rodes *et al.*, 2001); the use of personal care products (Conner *et al.*, 2001); and indoor chemical reactions (Weschler and Shields, 1997; Nazaroff and Weschler, 2004). Fibrous materials, pollen, mold spores and fragments, and tracked-in and blown-in soil particles are also components of indoor PM (Wallace, 1996a).

The contribution of outdoor PM to indoor PM concentrations can be substantial but highly variable. For residential buildings, the main entry routes of outdoor air are open windows and doors, cracks in the building shell, and mechanical ventilation systems such as swamp coolers and whole house fans. Investigators of a large, population-based study in California, the Particle Total Exposure Assessment Methodology Study (PTEAM Study) estimated that residential indoor PM<sub>10</sub>, on average, is roughly comprised of about 66% outdoor PM<sub>10</sub>; 75% for PM<sub>2.5</sub> (Ozkaynak *et al.*, 1996a,b). In a study of four Boston homes with air exchange rates below 1.0 air exchange per hour (ACH), Abt *et al.*, (2000) estimated that only 20-43 percent of indoor PM<sub>2</sub> to PM<sub>10</sub> were from outdoors, while 63-92 percent of indoor PM 0.02-0.3  $\mu\text{m}$  were from the outdoors. Abt *et al.*, (2000) and Long *et al.*, (2000) also found that the relative contribution of outdoor PM to indoor levels varied by particle size, with outdoor air generally contributing a majority of the smaller particles measured indoors, while indoor sources contributed more to the coarse (2-10 micrometers) fraction. Because these studies examined primarily older individuals who are less active in their homes than younger families may be, and were conducted on the

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east coast, the results of these studies may not reflect typical California proportions; however, they show that reductions in outdoor PM levels could have a major effect on indoor concentrations

Indoor combustion source emissions, such as those from smoking and cooking, are often intermittent and highly variable, but emissions can be very high, resulting in exposures that can have significant impacts on people's total daily exposure to PM (Long *et al.*, 2000). In the PTEAM Study homes with smokers, it was estimated that 30% of the PM<sub>2.5</sub> mass and 24% of the indoor PM<sub>10</sub> mass came from smoking. For homes in which cooking occurred during the monitoring period, 25% of the indoor PM<sub>2.5</sub> and PM<sub>10</sub> was estimated to come from the cooking activity (Ozkaynak, 1996b). These results are consistent with those of previous indoor studies that examined the impact of cigarette smoking on indoor PM levels, and they are consistent with subsequent studies of indoor cooking emissions that confirmed the high impact of cooking on indoor and personal PM levels (Abt *et al.*, 2000; Wallace, 2000b; Brauer *et al.*, 2000; Fortmann *et al.*, 2001).

For example, in an ARB-sponsored study, Fortmann *et al.* (2001) measured indoor and outdoor PM during 32 types of cooking activities with both gas and electric ovens and stovetops. Although concurrent outdoor levels reached only 20 µg/m<sup>3</sup>, indoor PM levels during and after cooking often exceeded 50 µg/m<sup>3</sup>, ARB's indoor air quality guideline level and ambient air quality standard for ambient PM<sub>10</sub> for 24 hours. Kitchen PM<sub>10</sub> levels exceeded more than 1400 µg/m<sup>3</sup> during frying, broiling, and baking activities with the gas stove (Fortmann *et al.*, 2001). Indoor PM levels during cooking with the electric stove were generally lower; however, cooking with the electric stove produced much higher indoor PM levels in two cases - frying tortillas and stovetop stir-frying. The highest concentrations of indoor PM were produced when using the self-cleaning cycle of the oven for several hours. Indoor PM<sub>10</sub> was over 3,600 µg/m<sup>3</sup> (over 2,000 µg/m<sup>3</sup> PM<sub>2.5</sub>) for the gas stove, and nearly 400 µg/m<sup>3</sup> PM<sub>10</sub> for the electric stove. Measurement of particle counts during cooking activities and oven cleaning indicated that particles were primarily smaller than 0.1 µm.

The burning of wood, candles, and incense can also be important combustion sources of residential indoor PM, especially in the 2.5 µm size range and below (Wasson *et al.*, 2002; Jetter *et al.*, 2002; Brauer *et al.*, 2000; Guo *et al.*, 2000; Lofroth *et al.*, 1991). Individual candles, for example, released 200-3600 µg/hr of PM<sub>10</sub>, and 100-1700 µg/hr of lead from the lead wick (Wasson *et al.*, 2002). Another investigator found a mean lead emission rate from candles of 770 µg/hr of lead (Alphen, 1999). Guo *et al.* (2000) modeled indoor PM<sub>2.5</sub> concentrations ranging from 4.3-1173 µg/m<sup>3</sup> based on PM emission measurements from candle burning. These indoor combustion sources produce PM with potentially harmful components similar to those from some outdoor PM combustion sources (Lofroth *et al.*, 1991).

Physical generation or re-suspension of particles also can contribute to airborne indoor PM levels. Soft or porous interior surfaces such as carpets and draperies have the potential to attract and re-emit particles (Thatcher and Layton, 1995; Kamens *et al.*, 1991). Particle concentrations can be high even in homes where good cleaning practices are used. The particles can become re-entrained in the indoor air when people walk or play (Wallace, 2000a; Roberts and Dickey, 1995; Abt *et al.*, 2000; Vette *et al.*, 2001).

Particles in house dust, such as metals, and semi-volatile chemicals such as pesticides and some PAHs that have their own toxic properties pose a risk to children (Rothenberg *et al.*, 1989; Roberts and Dickey, 1995; Lewis *et al.*, 1999; U.S. EPA, 1999a). Particles of house dust with these contaminants can be re-emitted to the air and subsequently inhaled, and may be ingested

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by children through hand-to-mouth behavior, often the primary route of exposure, or dermally absorbed when they spend time on the floor (Lewis *et al.*, 1994; Zartarian *et al.*, 1998; Zartarian and Leckie, 1998). For toxics such as lead, floor dust levels can be a major determinant of exposure.

Biological contaminants such as fungi, bacteria, house dust mites and pollen also contribute to indoor particle concentrations, especially in buildings with moisture problems from flooding or roof leaks that have not been properly repaired. Bioallergens, such as pollen, in outdoor air can also penetrate indoor spaces. Re-entrained road dust may be a particularly important source of bioallergens in both indoor and outdoor air (Miguel *et al.*, 1998).

### 2.2.3 Indoor and Personal PM Concentrations

Indoor PM concentrations sometimes exceed outdoor air concentrations, due to the presence of indoor sources of PM. For example, in one of the first comprehensive residential PM field studies, Spengler *et al.* (1981) found an increase in indoor concentrations of respirable PM of approximately 1  $\mu\text{g}/\text{m}^3$  per cigarette smoked per day, and about a 20  $\mu\text{g}/\text{m}^3$  increase per pack. Other examples are discussed in the Indoor PM Sources and Emissions Section above.

Additionally, people's personal exposures to PM sometimes exceed both indoor and outdoor concentrations, primarily because people tend to spend time very near pollutant sources, such as when cooking or cleaning. This has been called the "proximity effect", and reflects the fact that pollutant levels are highest near the source than farther away where emissions have become diluted in the air (McBride *et al.*, 1994). A small portion of the elevated personal exposure levels seen across PM studies also is attributed to the existence of a "personal cloud" of PM surrounding a person, due to re-suspension from clothing, the use of personal care products (Conner *et al.*, 2001), and skin flakes. Williams *et al.* (2000b) estimated a mean personal cloud of 3.1  $\mu\text{g}/\text{m}^3$ , and Rodes *et al.* (2001) estimated personal clouds of 3  $\mu\text{g}/\text{m}^3$  in two separate studies. However, Wallace (2000a) examined a breadth of studies and estimated an average personal PM<sub>10</sub> cloud of 30  $\mu\text{g}/\text{m}^3$ , ranging from 3-67  $\mu\text{g}/\text{m}^3$ . Personal PM<sub>2.5</sub> clouds were estimated to range from 6-27  $\mu\text{g}/\text{m}^3$ .

Consequently, because proximity to PM-emitting sources and activities can significantly increase people's actual exposures, most studies of indoor residential PM concentrations have included, or even focused on, measurement of occupants' personal exposures to PM. These have been obtained using personal samplers worn by study participants for one or more days as they go about their daily routines. These measurements provide a more accurate measure of people's exposure to PM, because PM levels are often higher very near people than at the location of indoor air sampling equipment in a room. Table 2.5 summarizes the major indoor and personal exposure PM studies conducted in the U.S. in recent years. Unlike earlier studies, there was little or no cigarette smoking in most of the studies listed in Table 2.5.

The first major study to measure indoor, outdoor, and personal PM concentrations in California was the PTEAM Study. Investigators measured PM<sub>10</sub> and PM<sub>2.5</sub> for 12-hour daytime and nighttime periods in 178 homes during the fall in Riverside, California. They found 12-hour daytime personal PM<sub>10</sub> concentrations to be about 50% higher than simultaneously measured daytime residential indoor or outdoor concentrations. Daytime personal concentrations averaged 150  $\mu\text{g}/\text{m}^3$ , while indoor and outdoor concentrations both averaged about 95  $\mu\text{g}/\text{m}^3$  (Clayton *et al.*, 1993, Ozkaynak *et al.*, 1996ab). Most importantly, 12-hour daytime personal PM<sub>10</sub> concentrations exceeded the California 24-hour ambient air quality standard level of 50  $\mu\text{g}/\text{m}^3$  for about 90% of the monitoring days, and exceeded the federal PM<sub>10</sub> standard level of

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150  $\mu\text{g}/\text{m}^3$  for 25% of the monitoring days. During nighttime, personal PM10 concentrations decreased and were similar to concurrent indoor and outdoor concentrations (roughly 80  $\mu\text{g}/\text{m}^3$ ), reflecting the influence of the proximity of people to PM sources during normal daytime activities in determining personal exposure concentrations.

Studies conducted since the PTEAM study in other locations and seasons have measured average personal PM10 concentrations from 11-68  $\mu\text{g}/\text{m}^3$ , and average personal PM2.5 concentrations ranging from 9-34  $\mu\text{g}/\text{m}^3$ . Average indoor concentrations of PM10 have ranged from 13-52  $\mu\text{g}/\text{m}^3$ , with indoor PM2.5 concentrations from 7-34  $\mu\text{g}/\text{m}^3$ . However, peak indoor levels have been high. For example, in a seven city study, Wallace *et al.* (2003) found that in all cities, at least 2% of all 1-hour measurements exceeded 1000  $\mu\text{g}/\text{m}^3$ . Long *et al.* (2000) measured indoor PM concentrations as high as 473  $\mu\text{g}/\text{m}^3$  in study homes. These elevated levels signify the presence of significant indoor source emissions for short periods of time relative to the average measurements.

Several studies have been conducted to examine the exposure of sensitive populations to PM. These have reported PM10 and PM2.5 exposures for subsets of individuals with chronic obstructive pulmonary disease (COPD), coronary heart disease, and asthma (Liu *et al.*, 2003; Williams *et al.*, 2000ac; Rojas-Bracho *et al.*, 2000; Linn *et al.*, 1999). Liu *et al.* (2003) found that mean personal PM2.5 concentrations were higher than indoor and outdoor concentrations for each of these three sensitive groups (as well as for healthy subjects) in Seattle, Washington, and that PM10 indoor concentrations were higher than outdoor concentrations for asthmatics. Williams *et al.* (2000ac) also found personal PM2.5 concentrations higher than indoor and outdoor concentrations in a Baltimore retirement facility in the winter of 1997. Rojas-Bracho *et al.* (2000) found that mean personal PM10 and PM2.5 concentrations were above indoor and outdoor PM10 and PM2.5 concentrations for 18 COPD patients in Boston.

Linn *et al.* (1999) monitored 15 COPD patients for PM2.5 exposures and 15 patients for PM10 exposures in Los Angeles during the fall and winter. Unlike other studies, this study found personal concentrations similar to both those indoors or outdoors. The authors suggest that the lack of increased personal PM in these subjects having severe COPD may be due to reduced personal activity, less time spent outside of the home, and other reasons. The pooled correlation of personal PM concentrations to ambient concentrations at a monitoring station were quite low.

A recently completed study by Suh (2003) has increased our understanding of Californians' exposures to PM in a sensitive subpopulation. The investigators examined the relationships among outdoor and indoor concentrations and personal exposures across different seasons. They examined daily PM exposures of a group of 15 individuals with COPD in Los Angeles over seven sequential days in the summer-fall and/or winter. Personal, indoor, and outdoor PM2.5,  $\text{NO}_3^-$  and elemental carbon (EC) concentrations varied by season, with the exception of outdoor  $\text{NO}_3^-$ . For winter and summer-fall PM levels, respectively, the personal PM2.5 means (19.6, 25.1  $\mu\text{g}/\text{m}^3$ ) and maxima (63.5, 137.8  $\mu\text{g}/\text{m}^3$ ) were higher than the indoor means (16.9, 18.1  $\mu\text{g}/\text{m}^3$ ) and maxima (49.5, 94.8  $\mu\text{g}/\text{m}^3$ ) and outdoor means (13.5, 19.3) and maxima (56.5, 53.5  $\mu\text{g}/\text{m}^3$ ). For  $\text{NO}_3^-$  and EC, higher outdoor (2.8-3.1  $\mu\text{g}/\text{m}^3$ ), as compared to indoor (1.1-1.7  $\mu\text{g}/\text{m}^3$ ) and personal (1.2-1.6  $\mu\text{g}/\text{m}^3$ ) levels, were found in both seasons, reflecting the fact that motor



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**Table 2.5. Summary of Recent Indoor PM Exposure Studies (means)**

LOCATION	GROUP	YEAR	SEASON	PM 2.5 means - $\mu\text{g}/\text{m}^3$						REFERENCE
				PERSONAL	(n)	INDOOR	(n)	OUTDOOR	(n)	
Los Angeles, CA	COPD Subjects	2001-2002	multiple	17.7 $\pm$ 11.9	91	17.6 $\pm$ 11.4	106	28.8 $\pm$ 20.4	103	Suh 2004
Raleigh, NC	African Americans	2000-2001	multiple	23.0 $\pm$ 16.1	712	19.3 $\pm$ 8.4	761	19.3 $\pm$ 8.4	761	Williams 2003b
Seattle, WA	COPD Subjects	2000-2001	multiple	10.5 $\pm$ 7.2	307	8.5 $\pm$ 5.1	443	9.2 $\pm$ 5.1	437	Liu 2003
	Healthy Subjects			9.3 $\pm$ 8.4	183	7.4 $\pm$ 4.8	193	9.0 $\pm$ 4.6	194	
	Asthmatics			13.3 $\pm$ 8.2	263	9.2 $\pm$ 6.0	276	11.3 $\pm$ 6.4	272	
	Coronary Heart Dis			10.8 $\pm$ 8.4	325	9.5 $\pm$ 6.8	329	12.6 $\pm$ 7.9	323	
Boston, MA	Asthmatic Children	2000	multiple			12.3 <sup>1</sup>	NR	9.0 <sup>1</sup>	NR	Brugge 2003
						33.8 <sup>2</sup>	NR	13.1 <sup>2</sup>	NR	
Los Angeles, CA	COPD Subjects	2000	winter	19.6 $\pm$ 14.5	87	16.9 $\pm$ 11.7	92	13.5 $\pm$ 8.5	92	Suh 2003
		1999	summer-fall	25.1 $\pm$ 20.8	92	18.1 $\pm$ 11.1	97	19.3 $\pm$ 9.0	96	
Detroit, MI	Asthmatic Children	1999-2000	multiple			34.4 $\pm$ 21.7	362	15.6 $\pm$ 8.2	NR	Keeler 2002
Fresno, CA	Retirement Facility	1999	winter	13.3	24	9.7	24	20.5	28	Evans 2000
			spring	11.1	12	8.0	24	10.1	28	
Baltimore, MD	Elderly	1999	winter	19				5.6		Sarnat 2000
		1997	summer	27				25		
Baltimore, MD	Retirement Facility	1998	summer	13.0 $\pm$ 4.2	23	10.0 $\pm$ 4.7	16	22.0 $\pm$ 12.0	28	Williams 2000c
		1997	winter	34.4 [PM <sub>1.5</sub> ]	NR	17.4	NR	17.0 [PM <sub>1.5</sub> ]	NR	Williams 2000a
Boston, MA	Residential Homes	1998	multiple			11.9 $\pm$ 9.6	211	11.1 $\pm$ 6.8	210	Long 2000
		1996	multiple			13.9 $\pm$ 15.2	63	11.7 $\pm$ 6.5	64	Abt 2000
Birmingham, AL	Residential Homes	1997-1998	summer	18.6 $\pm$ 6.4	30	16.1 $\pm$ 9.6	30	26.5 $\pm$ 9.5	30	Lachenmeyer 2000
			winter	10.0 $\pm$ 3.3	30	11.2 $\pm$ 5.4	30	12.2 $\pm$ 5.1	30	
Boston, MA	Residential Homes (people with COPD)	1996-1997	winter	21.6 $\pm$ 15.2	93	17.2 $\pm$ 13.0	93	10.9 $\pm$ 9.2	94	Rojas-Bracho 2000
			summer	21.5 $\pm$ 11.9	131	17.7 $\pm$ 14.9	138	16.4 $\pm$ 13.0	138	
Los Angeles	Elderly w/ COPD	1996-1997	winter	24		25		25		Linn 1999
7 U.S. Cities	Asthmatic Children	NR	NR			27.7 $\pm$ 35.9	448 0	13.6 $\pm$ 7.5	403 1	Wallace 2003a
United States	Office Buildings	1994-1998	summer-winter			7.2	453	14.7	453	Burton 2000

LOCATION	GROUP	YEAR	SEASON	PM 10 means - $\mu\text{g}/\text{m}^3$						REFERENCE
				PERSONAL	(n)	INDOOR	(n)	OUTDOOR	(n)	
Raleigh, NC	African Americans	2000-2001	multiple			27.7 $\pm$ 19.6	761	30.4 $\pm$ 14.1	761	Williams 2003b
Seattle, WA	COPD Subjects	2000-2001	multiple			14.1 $\pm$ 6.6	437	14.3 $\pm$ 6.8	435	Liu 2003
	Healthy Subjects					12.6 $\pm$ 7.8	206	14.5 $\pm$ 7.0	200	
	Asthmatics					19.4 $\pm$ 11.1	274	16.4 $\pm$ 7.4	269	
	Coronary Heart Dis					16.2 $\pm$ 11.3	324	18.0 $\pm$ 9.0	324	
Los Angeles, CA	COPD Subjects	2000	winter	35.0 $\pm$ 22.0	89	30.6 $\pm$ 21.2	95	36.1 $\pm$ 13.2	94	Suh 2003
		1999	summer-fall	29.1 $\pm$ 12.8	19	29.0 $\pm$ 14.7	21	15.2 $\pm$ 8.6	21	
Detroit, MI	Asthmatic Children	1999-2000	multiple	68.4 $\pm$ 39.2	252	52.2 $\pm$ 30.6	363	25.8 $\pm$ 11.8	NR	Keeler 2002
Fresno, CA	Retirement Facility	1999	winter			15.1	24	28.2	28	Evans 2000
			spring	37.3	12	16.7	24	28.7	28	
Baltimore, MD	Elderly	1999	winter	28				7.5		Sarnat 2000
		1997	summer	34				34		
Baltimore, MD	Retirement Facility	1998	summer			13.5 $\pm$ 5.9	15	30.0 $\pm$ 13.7	28	Williams 2000c
Boston, MA	Residential Homes	1998	multiple			19.4 $\pm$ 12.7	212	12.7 $\pm$ 7.5	107	Long 2000
		1996	multiple			19.6 $\pm$ 16.1	64	17.1 $\pm$ 9.1	64	Abt 2000
Boston, MA	Residential Homes (people with COPD)	1996-1997	winter	40.7 $\pm$ 26.8	93	37.3 $\pm$ 23.2	93	18.5 $\pm$ 15.9	95	Rojas-Bracho 2000
			summer	34.7 $\pm$ 17.5	132	28.3 $\pm$ 25.4	138	24.8 $\pm$ 21.9	137	
Los Angeles	Elderly w/ COPD	1996-1997	winter	35		33		40		Linn 1999
Riverside, CA	Residential Homes	1996	fall	150	NR	95	NR	95	NR	Ozkaynak 1996
United States	Office Buildings	1994-1998	summer-winter			11.4	588	23.1	588	Burton 2000

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vehicles are their major source. In addition, the loss of  $\text{NO}_3^-$  may occur indoors due to its dissociation of particulate nitrate back to gaseous form under conditions of increasing temperature and decreasing humidity.  $\text{NO}_3^-$  and EC comprised a small fraction of personal, indoor, and outdoor  $\text{PM}_{2.5}$  (max. 28.5% and 17%, respectively). The indoor PM levels were low compared to those measured in studies of the general population, consistent with the subjects' limited personal activity and very little time spent near smoking, cooking, vehicles, or other major PM sources. Personal PM was highly correlated with indoor PM, and indoor and outdoor PM correlations were significant as well. Statistical modeling results indicated that significant predictors of higher personal PM were time near ETS, location near a major road, higher population density, cooking activity, and location in an inland area (vs. coastal).

A companion study to the above study by Suh and Koutrakis (2004) used a similar design for 16 healthy persons in Los Angeles during the summer and/or winter. The study also involved more detailed characterizations of the PM levels and ventilation characteristics of the subject's homes than the COPD study. Both personal and indoor  $\text{PM}_{2.5}$  averaged approximately  $18 \mu\text{g}/\text{m}^3$  (range 2 -  $68 \mu\text{g}/\text{m}^3$ ). Outdoor  $\text{PM}_{2.5}$  levels were generally much higher (mean  $29 \mu\text{g}/\text{m}^3$ , range 5 -  $103 \mu\text{g}/\text{m}^3$ ) than corresponding indoor and outdoor concentrations in both seasons. Outdoor nitrate levels averaged  $11 \mu\text{g}/\text{m}^3$  (range 0.3 -  $55 \mu\text{g}/\text{m}^3$ ), and were also higher than indoor and personal levels. In contrast, outdoor EC levels were similar to corresponding indoor and personal levels, averaging  $2 \mu\text{g}/\text{m}^3$  (range 0 -  $7 \mu\text{g}/\text{m}^3$ ). Personal exposures to these three particulate measures were more strongly associated with indoor concentrations as compared to outdoor concentrations, which may be attributed to the fact that the subjects spent a majority of their time indoors at home. The investigators found that indoor  $\text{PM}_{2.5}$  was the largest contributor to personal  $\text{PM}_{2.5}$  levels, accounting for 65-100% of daily personal  $\text{PM}_{2.5}$ , on average. Significant individual and diurnal variations in all PM measurements, air exchange rates, and PM-generating activities such as cooking and cleaning were also observed.

Far fewer studies have been conducted in public buildings than in residences. Indoor PM concentrations in public and commercial buildings appear to often be lower than ambient concentrations. Lower indoor PM concentrations in public and commercial buildings are due to the use of particle filters in mechanical ventilation systems, un-openable windows, and the lack of many indoor sources typically present in residences. As part of the U.S. EPA's Building Assessment Survey and Evaluation (BASE) study, Burton *et al.* (2000) reported PM concentrations from 100 randomly selected office buildings throughout the United States. Mean indoor  $\text{PM}_{2.5}$  and  $\text{PM}_{10}$  concentrations were  $7.2 \mu\text{g}/\text{m}^3$  and  $11.4 \mu\text{g}/\text{m}^3$ , respectively, while outdoor levels were higher, at  $14.7 \mu\text{g}/\text{m}^3$  and  $23.1 \mu\text{g}/\text{m}^3$ , respectively. However, as with residences, the presence of indoor sources in public and commercial buildings can produce indoor concentrations that exceed concurrent ambient concentrations, especially if smoking is allowed in the building. For example, in a study of 38 commercial buildings in the Pacific Northwest, Turk *et al.* (1987) found that buildings where smoking was permitted had average indoor PM levels 3.5 times higher than concurrent outdoor levels and indoor levels in buildings where smoking was prohibited. Sheldon *et al.* (1988) measured indoor PM in six buildings in the eastern U.S., and found indoor PM concentrations generally lower than outdoors where there was no smoking, but much higher indoor concentrations where smoking was allowed.

Lillquist *et al.* (1998) reported indoor and outdoor  $\text{PM}_{10}$  measurements in three Utah hospitals over one winter season. Significant variability in indoor PM levels was found both among room types and among hospitals, and the relationship between indoor  $\text{PM}_{10}$  levels and outdoor levels was highly variable. The ICUs had significantly lower  $\text{PM}_{10}$  levels than other types of rooms, after adjusting for hospital differences. Thus, the most critically ill individuals may experience

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some protection in hospitals from ambient PM; however, in general, hospitals do not offer protection from ambient PM.

### 2.2.4 Carbon Monoxide

Carbon monoxide (CO) is a colorless, odorless, tasteless gas. It is a product of incomplete combustion, emitted from sources such as vehicles (in exhaust), gas and propane appliances, woodstoves, kerosene heaters, and cigarettes. CO can trigger acute health effects, even death, at very high levels, or flu-like symptoms and other effects at lower levels over longer periods of time.

CO is regulated in the ambient environment, but not indoors. However, transient elevated concentrations in outdoor places such as tunnels and parking garages are not widely regulated. The state of California has an 8-hour average ambient air quality standard of 9.0 ppm (10 mg/m<sup>3</sup>) and a one-hour standard of 20 ppm (23 mg/m<sup>3</sup>). These are also ARB's recommended indoor guideline levels for CO. In one large California study, about 5% of homes were found to have CO levels that exceeded the State 8-hour ambient air quality standard for CO (Wilson *et al.*, 1993).

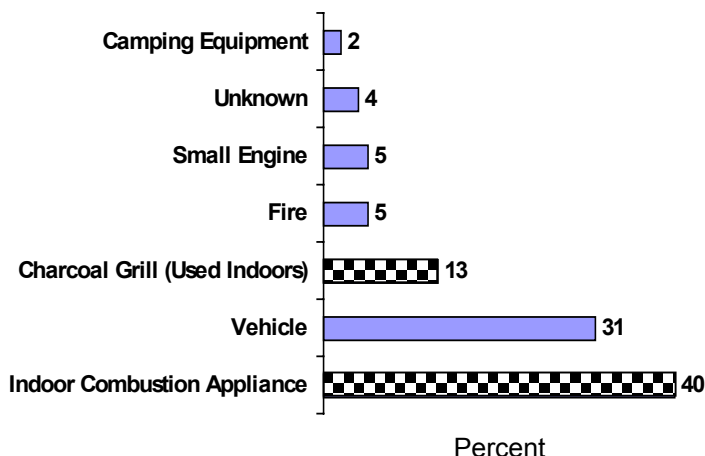
#### 2.2.4.1 Death

The acute health effects of CO exposure have been well documented in multiple studies since the 1970s. Acute CO poisoning results from a lack of oxygen in the bloodstream due to formation of a CO-hemoglobin complex (carboxyhemoglobin) that prevents oxygen from binding to hemoglobin. Symptoms of acute poisoning include headache, nausea, lethargy and inability to concentrate, unconsciousness, and death at very high concentrations. An estimated 600 deaths per year in the United States throughout the 1990s were attributable to unintentional CO poisoning (cited in Raub *et al.*, 2000).

A California study of ten years of death certificates showed that about 30 - 40 deaths occur in California each year, on average, due to unintentional carbon monoxide (CO) poisoning (Girman *et al.*, 1998; Liu *et al.*, 1993a, 2000). About two-thirds of those deaths were attributable to indoor sources. As shown in Figure 2.2, the indoor sources most implicated in the CO poisonings were combustion appliances (usually malfunctioning or poorly tuned) and charcoal grills and hibachis used indoors. Together these accounted for over 50% of the deaths. The types of cooking and heating appliances

included in the indoor combustion appliance category were wall heaters (37%), free-standing heaters (19%), stoves (16%), water heaters (9%), furnaces (9%), and floor heaters (7%).

**Figure 2.2: Causes of CO Deaths in California**



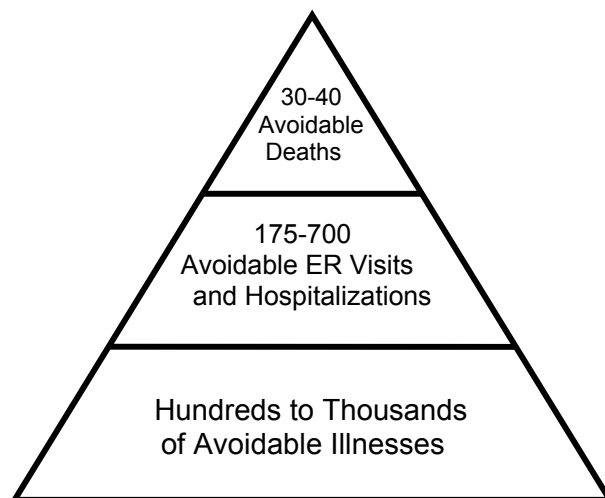
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Natural gas was associated with 75% of the deaths, propane with 24%, and kerosene with less than 1%. Motor vehicles, such as those left running in an attached garage, also took a substantial toll.

### 2.2.4.2 Non-lethal Health Effects

In an examination of 1991-1994 California CO deaths and hospitalization discharge data, Waldman (1996) found that about 3 to 7 times as many hospitalizations for non-fatal CO poisoning occurred as did deaths from CO poisoning. This totals about 100-300 documented hospitalizations for an average year. However, uncertainties in the patient discharge database and the omission from the database of emergency room discharges that did not result in hospitalization led the investigator to conclude that the actual number of hospitalizations attributable to CO poisoning was at least several times higher. From the pertinent literature, ARB staff and Waldman estimate that five to twenty times (175-700) as many individuals as die from accidental CO poisoning are treated in emergency rooms or hospitalized each year due to serious, non-fatal CO poisonings, and that hundreds to thousands more suffer from undiagnosed heart problems, headache, flu-like symptoms, and other illnesses attributable to CO exposure (Cook *et al.*, 1995; Mah, 2000; Kirkpatrick, 1987; MMWR, 1982; Waldman, 1996). Figure 2.3 illustrates the likely magnitude of these non-lethal effects on the population.

Recent studies have further documented chronic health effects following acute exposures and those due to prolonged exposures to CO. These chronic effects include lethargy, headaches, concentration problems, amnesia, dementia, psychosis, Parkinsonism, memory impairment,



**Figure 2.3: Annual California CO Cases**

personality alterations, signs of parietal dysfunction, and other minor symptoms (Townsend and Maynard, 2002; Mathieu-Nolf, 2002). In a study of cardiovascular hospital admission data in Los Angeles between 1992 and 1995, Linn *et al.*, (2000) found that CO showed the most consistently significant relationship among the pollutants considered, and concluded that a wintertime increase in CO of 1.1 to 2.2 ppm predicted an increase of 4% in cardiovascular

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admissions (~20 extra admissions per day). The elderly are especially susceptible to chronic effects of CO.

Children are especially susceptible to harm from CO. Because they inhale more air per unit of body weight than do adults at similar activity levels, children will inhale a higher dose proportionately than adults, and will experience symptoms more quickly than adults. The unborn appear to be susceptible to adverse effects after exposure to CO as well. In a study of the relationship between ambient air pollution and low birth weight in the northeastern U.S., Maisonet *et al.* (2001) observed increased odds of low birth weight for every 1 ppm increase in CO during the third trimester for the entire population, and a similar increased risk in the African-American population across all three trimesters. In an unrelated study, Ritz *et al.* (2000) estimated that the risk of pre-term birth increases by 12% per 3 ppm increase in CO averaged over six weeks before birth, and by 4% averaged over the first month of pregnancy. Ritz *et al.* (2000) also found an increased risk of heart defects with increased ambient CO exposure during the second trimester of pregnancy. However, attributing the effects strictly to CO is difficult due to the presence of other correlated pollutants. Because these effects were seen with small increases in CO, the presence of indoor sources of CO in homes with pregnant women is a serious concern.

### 2.2.4.3 Carbon Monoxide Emissions and Concentrations

Most homes have relatively low CO levels, except for short intermittent elevations during use of an indoor source. Indoor CO concentrations can increase rapidly when a highly emitting source is present in an enclosed environment. For example, Jetter *et al.* (2002) measured CO emission rates from incense burning from 159-531 mg/hr, resulting in an estimated peak CO concentration of 9.6 mg/m<sup>3</sup> in a modeled typical room in a home. This is just at the California 8-hour standard level of 9 ppm (10 mg/m<sup>3</sup>); in combination with outdoor CO and any additional indoor sources, this home would have exceeded the standard. Pelham *et al.* (2002) reviewed CO levels in indoor ice arenas, stressing that U.S. and global CO exposure problems remain in these locations (only three U.S. states regulate CO levels in ice arenas).

In a study of 277 Californian homes in 1992, Wilson *et al.* (1993) measured indoor and outdoor CO levels. Thirteen homes had indoor 8-hour CO concentrations above the California 8-hour outdoor standard and indoor air quality guideline of 9 ppm. Several homes had indoor CO 1-hour values greater than the state standard of 20 ppm; in one case this was attributed to the (dangerous) use of gas burners for residential heating. In a second California study (focused on PAHs) of 280 homes in northern California, only two homes exceeded California CO standard levels, one due to use of a fireplace and the other from gas heat (Sheldon *et al.*, 1993). However, in many homes, short-term excursions up to 42 ppm were observed, such as when a gas space heater was turned on.

CO levels in well-maintained indoor environments where appliances are operated properly tend to be relatively low. In surveys of 136 'non-problem' buildings in New York City between 1997 and 1999, Springston *et al.* (2002) measured indoor CO concentrations ranging from 0.2 to 10.3 ppm; only 0.04% of all readings exceeded 10 ppm.

A limited number of studies have shown that infiltration of vehicle exhaust emissions into residences can increase the indoor concentration of CO within those residences. Wilson *et al.* (1993) found that the presence of an attached garage was a significant factor related to elevated indoor concentrations of CO versus outdoor concentrations. Gamage *et al.* (1994) found that the CO concentration in the bedroom located above a garage increased from less

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than 1 ppm to 17 ppm after a garaged vehicle's engine was operated for a period of 3 minutes. In a detailed Canadian study, Graham *et al.* (2004) measured the concentration of CO inside 16 residences over two seasons while a vehicle in the attached garage was operated under brief cold-start and hot-soak conditions. Significant net changes in indoor CO concentrations were observed to be associated with the vehicle cold start, but not the hot soak running condition.

### 2.2.5 Nitrogen Dioxide and Associated Acids

Nitrogen dioxide (NO<sub>2</sub>) is a red to dark brown gas with a pungent acrid odor. It is emitted from combustion sources such as natural gas and propane-fueled appliances, wood burning stoves and fireplaces, kerosene heaters, charcoal grills and motor vehicles. Adverse health effects attributable to NO<sub>2</sub> include exacerbation of asthma (especially in children), respiratory symptoms and infection, lung damage, and lung disease after long periods of exposure. Several nitrogen compounds related to NO<sub>2</sub> are found in indoor environments; these include nitrous acid (HONO) and nitric oxide (NO). NO<sub>2</sub> is the only nitrogen oxide regulated as a pollutant in outdoor air. California has a one-hour ambient air quality standard for NO<sub>2</sub> of 0.25 ppm, not to be exceeded; this also serves as an indoor air quality guideline. The national ambient air quality standard is an annual mean of 100 µg/m<sup>3</sup> (0.053 ppm). Based on reports that 20-30% of the population use their gas stove (despite clear warnings against this) for space heating (Phillips *et al.*, 1990), indoor NO<sub>2</sub> is estimated to exceed the State ambient air quality standard at times in 10-30% of California homes.

#### 2.2.5.1 Health Effects of Nitrogen Dioxide

A number of studies have shown that nitrogen dioxide (NO<sub>2</sub>) exposure can result in detrimental effects in the lung. Investigators using human clinical studies have recently reported on NO<sub>2</sub> and its effects on airway antioxidant status, inflammatory cell and mediator responses, and lung function (Becker and Soukup, 1999; Barck *et al.*, 2002; Blomberg *et al.*, 1999; Delvin *et al.*, 1999). Data from Delvin *et al.* (1999), for example, demonstrate that 2 ppm NO<sub>2</sub> can induce a mild inflammatory response in the airways of healthy adults, and that NO<sub>2</sub> may cause a mild impairment of lung antibacterial capacity. This study suggests that possible increases in viral clinical symptoms associated with NO<sub>2</sub> may result from effects of the NO<sub>2</sub> on host defenses that normally prevent the spread of virus. Data from European studies indicate that NO<sub>2</sub> is a pro-inflammatory air pollutant under conditions of repeated exposure at a relatively high concentration of 2 ppm, 4 hours per day, for 4 days (Blomberg *et al.*, 1999). However, brief exposures (less than 1 hour) to ambient concentrations of NO<sub>2</sub> can enhance allergic inflammatory reaction in the airways of asthmatics (Barck *et al.*, 2002).

As discussed earlier, the NAS Institute of Medicine's Committee on the Assessment of Asthma and Indoor Air determined that there is sufficient scientific evidence to conclude that high levels of indoor NO<sub>2</sub> can exacerbate asthma (IOM, 2000). A recent epidemiology study conducted in Australia (Pilotto *et al.*, 2003) supports this finding and concludes that asthma symptoms were reduced in primary school children after intervening to remove a high-NO<sub>2</sub>-production source at school.

Evidence suggests an association between exposure to NO<sub>2</sub> and increased respiratory symptoms in children. Neas *et al.* (1991) studied the effect of indoor NO<sub>2</sub> on respiratory symptoms in 1,567 children aged 7-11 in six U.S. cities from 1983 to 1988. Analysis of symptoms obtained through a questionnaire indicate that a 15 ppb increase in annual (average) indoor NO<sub>2</sub> was associated with an increased cumulative incidence of lower respiratory

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symptoms. Girls showed a stronger association (OR = 1.7) than did boys (OR = 1.2). There was not an association between pulmonary function and NO<sub>2</sub> levels.

Chauhan *et al.* (2003) examined the relationships between NO<sub>2</sub> exposure and asthma severity in 8-11 year old children during a respiratory viral infection. Investigators concluded that exposure to 7-day average NO<sub>2</sub> levels of about 11 ppb before the start of a respiratory viral infection is associated with an increase in the severity of virus-induced asthma exacerbations.

California investigators also have reported health effects of NO<sub>2</sub> exposure on children. Peters *et al.* (1999a) studied school children to assess respiratory effects due to long-term exposure to four pollutants: ozone, particulate matter, acids, and NO<sub>2</sub>. Peters found NO<sub>2</sub> significantly associated with reduced lung function in female children within a cohort of 3,292 school children in twelve Southern California communities. Increases in bronchitis symptoms of children with pre-existing asthma also were associated with increases in ambient NO<sub>2</sub> levels (McConnell *et al.*, 2003). It is important to note in the McConnell *et al.*, study the annual average concentration of NO<sub>2</sub> was about 19 ppb, well below the national annual average standard of 53 ppb. Within a cohort of 846 asthmatic children residing in eight urban areas of the U.S., Mortimer *et al.* (2002) found a 48% increase in the likelihood of asthma symptoms associated with an increase in the 6-day average NO<sub>2</sub> levels. Within a panel of 138 children in central Los Angeles, Ostro *et al.* (2001) found increased odds for shortness of breath and wheezing associated with a 50 ppb increase in the 1-hour maximum NO<sub>2</sub> concentrations.

Aggregate results from numerous individual studies dealing with exposure to NO<sub>2</sub> and respiratory illness in children have been inconclusive. However, Hasselblad *et al.* (1992) conducted a meta-analysis on studies with inconsistent results to conclude that children exposed to a long-term increase of 30 µg/m<sup>3</sup> NO<sub>2</sub> (approximately 16 ppb) have about a 20% increase in the odds for developing respiratory illness.

Investigators have also identified a relationship between women (age 20-44) in England who use gas appliances and develop asthma-like symptoms (Jarvis *et al.*, 1996). Women who primarily used gas cooking appliances (known to emit NO<sub>2</sub>) had an increased risk for asthma attacks, wheeze, and waking with shortness of breath. The women who used a gas stove also had reduced lung function and increased airway obstruction compared to women who did not use gas stoves. These associations were not observed in men, possibly because they did not experience the high concentrations of pollutants near the cooking source (according to the authors).

### 2.2.5.2 Nitrogen Dioxide Sources, Emissions, and Concentrations

Nitrogen dioxide is emitted during combustion; sources include motor vehicles, tobacco smoke, and combustion appliances such as gas kitchen stoves, gas, propane, and kerosene-fueled heaters, wood burning stoves, fireplaces, and charcoal grills. In the absence of indoor sources, indoor NO<sub>2</sub> levels are influenced by outdoor levels due to the infiltration of outdoor air (Spengler *et al.*, 1994b; Weschler and Shields, 1994; Levy *et al.*, 1998). When indoor combustion sources such as wall furnaces, floor furnaces, gas stoves, and unvented gas logs (not permitted in California) are present, they have a large influence on indoor NO<sub>2</sub> concentrations (Spengler *et al.*, 1994b; Pitts *et al.*, 1989; Wilson *et al.*, 1986; Wilson *et al.*, 1993).

Several oxidized nitrogen compounds in addition to NO<sub>2</sub> are emitted during combustion by gas appliances and/or are formed through chemical reactions. The most notable additional species, are nitric oxide (NO), nitrous acid (HONO), and nitric acid (HNO<sub>3</sub>) (Spicer *et al.*, 1993). Pitts *et*

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*al.*, (1985) reported direct observation of the formation of gaseous HONO from the reaction of NO<sub>2</sub> (at ppm levels) with water vapor in indoor environments. Based on removal reactions, indoor NO<sub>2</sub> has a lifetime of about one hour, while the lifetime for NO and HONO is several hours (Spicer *et al.*, 1993). HONO can be retained on indoor surfaces for extended periods, then be released causing elevated concentrations after a combustion appliance is no longer in use (Spicer *et al.*, 1993; Febo and Perrino, 1991). HONO concentrations in homes with gas appliances are typically greater indoors than outdoors and range from 10-20 ppb (Febo and Perrino, 1991; Spengler *et al.*, 1993). HONO is present in indoor air as an acidic aerosol and is likely to be a respiratory irritant, though its respiratory toxicity has not been thoroughly investigated.

Nitrogen dioxide is the most prevalent of the nitrogen oxides and has been the focus of numerous emission and indoor concentration studies. In an ARB-funded cooking study, Fortmann *et al.* (2001) measured indoor NO<sub>2</sub> during various cooking protocols. Measurement periods varied from approximately 1 to 5 hours, representing food preparation, cooking, and clean-up times. Nitrogen dioxide levels increased when a gas stove was used for cooking. For example, while making a fried chicken dinner, average indoor NO<sub>2</sub> levels reached 400 ppb. Other cooking tasks such as broiling fish, baking lasagna, frying tortillas, and stir-frying produced average indoor NO<sub>2</sub> levels ranging from 30 to 170 ppb. During a cycle of automatic oven cleaning with a gas stove, average indoor NO<sub>2</sub> levels exceeded 400 ppb. NO<sub>2</sub> concentrations remained below 45 ppb during the cooking protocols performed with an electric stove and range.

Indoor NO<sub>2</sub> levels can reach unhealthy indoor levels in some situations. In the California Residential Indoor Air Quality Study, indoor NO<sub>2</sub> levels were measured up to 177 ppb as 48-hour averages (Wilson *et al.*, 1993). At this level, it is very likely that the 250 ppb California one-hour standard was exceeded for at least some portion of the time. Dennekamp *et al.* (2001) measured 5-minute peaks up to 1000 ppb NO<sub>2</sub> when cooking with a 4-burner gas stove (measured at face level in front of the cook). In a Boston study, Brugge *et al.* (2003) found indoor NO<sub>2</sub> levels were either close to or exceeded the NAAQS annual level of 53 ppb.

When investigators collect samples over several days, reported concentrations are lower than peak concentrations measured during a distinct exposure event. For example, Lee *et al.* (2002) measured indoor NO<sub>2</sub> and HONO levels in 119 residences in southern California over a 6-day sampling period. The average indoor and outdoor concentrations of NO<sub>2</sub> were 28 and 20.1 ppb, respectively. Zipprich *et al.* (2002) collected 48-hour passive NO<sub>2</sub> samples in Richmond, Virginia. Mean concentrations in the bedrooms, living rooms, and outdoors were 18, 19, and 15 ppb, respectively. 98% of the homes had gas stoves.

Spengler *et al.* (1994b) measured personal exposures, as well as indoor and outdoor levels of NO<sub>2</sub>, for about 700 individuals in the Los Angeles basin. Passive samples were collected over 24- or 48-hour periods. The median personal and outdoor levels were 35 ppb while the median indoor level was 24 ppb. However, the contribution of gas appliances and gas pilot lights to total exposure was evident. "Personal exposures for those in homes with gas ranges with pilot lights average 10 ppb greater than those with electric ranges, and 4 ppb greater than those with gas ranges without pilot lights" (Spengler *et al.*, 1994b).

Levy *et al.* (1998) identified the use of a gas stove in a home as the most significant contributor to personal NO<sub>2</sub> exposure. Their study, conducted in 15 countries found that mean personal (2-day average) NO<sub>2</sub> exposure was 34.8 ppb in homes with a gas stove that was used during the sampling period, compared to 20.5 ppb in homes without gas stove use.



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### 2.2.6 Ozone

Ozone (O<sub>3</sub>), the primary component of smog, is an invisible, yet highly corrosive, odorous, and chemically reactive gas. Ozone is chemically unstable, and so it breaks down or reacts with many surfaces, liquids, and chemicals. Ozone is typically higher outdoors than indoors. It becomes elevated indoors most commonly from the infiltration of outdoor ozone through doors, windows, and swamp coolers, and sometimes from direct emissions indoors by devices such as certain types of copy machines, laser printers, and “air purifiers”.

#### 2.2.6.1 *Health Effects of Ozone*

Ozone is an oxidizing pollutant and strong irritant that attacks the respiratory system, leading to the damage of lung tissue. Exposure to ozone damages the alveoli, the individual air sacs in the lung where the exchange of oxygen and carbon dioxide between the air and blood takes place (ARB/OEHHA, 2004). Increased occurrence of respiratory symptoms, such as cough, pain on deep breath, and difficulty taking a deep breath are associated with exposure to ozone (Schelegle and Adams, 1986; McDonnell *et al.*, 1999; Kulle *et al.*, 1985; Folinsbee *et al.*, 1977; Seal *et al.*, 1993). Ozone also induces cellular and biochemical changes indicative of lung inflammation (Devlin *et al.*, 1991, 1996; Balmes *et al.*, 1996; Aris *et al.*, 1993). A major result from the Children’s Health Study indicates that children living in high ozone communities who actively participate in several sports are three times more likely to develop asthma than children in these communities not participating in sports or those that live in low ozone communities (Gauderman *et al.*, 2000).

Epidemiological studies have found statistically significant associations between outdoor O<sub>3</sub> concentrations and various adverse health impacts, including increased asthma symptoms (Wittemore and Korn, 1980; Thurston *et al.*, 1997; Delfino *et al.*, 1996; Mortimer *et al.*, 2002), increased respiratory symptoms (e.g., Brunekreef *et al.*, 1994), and reduced lung function (Brunekreef *et al.*, 1994; Brauer *et al.*, 1996). Some studies also report statistically significant associations between O<sub>3</sub> and hospital admissions or emergency room visits, primarily for asthma or other respiratory causes including COPD and bronchitis (Sheppard *et al.*, 1999; Schwartz, 1995; Delfino *et al.*, 1997, 1998; Burnett *et al.*, 1997; Anderson *et al.*, 1997). Ozone has also been linked to increased school absenteeism for respiratory illnesses (Gilliland *et al.*, 2001), and reduced lung function growth in children (Frischer *et al.*, 1999).

California has an ambient air quality standard for O<sub>3</sub> of 0.09 ppm for one hour, while the federal standard is 0.08 ppm averaged over eight hours. Both standards are currently under review, and California has proposed an 8-hour standard of 0.070 ppm (ARB/OEHHA 2005).

#### 2.2.6.2 *Indoor Sources and Concentrations of Ozone*

Outdoor air is the most common source of indoor ozone (Weschler *et al.*, 1989). Outdoor ozone (a component of smog; formed by the photochemical reaction of volatile organic compounds and nitrogen oxides emitted primarily by motor vehicles and industries) enters homes through doors, windows, and numerous air leaks in buildings and their ventilation systems. Studies have shown that indoor ozone levels generally follow the diurnal and seasonal patterns of outdoor ozone, with higher levels in the daytime and summer months (Liu *et al.*, 1993b; Weschler *et al.*, 1994; Liu *et al.*, 1995; Avol *et al.*, 1998; Geyh *et al.*, 2000; Lee *et al.*, 2002). Like outdoor concentrations, indoor ozone levels can also remain elevated for long periods of time (eight hours or more), and display peak variations throughout the day (Weschler *et al.*, 1989). Indoor ozone levels typically range from 20 to 80% of outdoor ozone levels (Weschler *et al.*, 1989).

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Using a swamp cooler or whole-house fan on a high-ozone day can increase air exchange rates enough to produce indoor ozone levels very close to outdoor levels for hours at a time (Avol *et al.*, 1996).

The most common indoor sources of O<sub>3</sub> are poorly maintained laser printers and photocopiers, and O<sub>3</sub> generating-devices that are marketed as various types of room deodorizers and air cleaners. In particular, ozone generators that are marketed as “air purifiers” have been found to produce hazardous levels of indoor ozone. Several studies have shown that ozone at levels produced by ozone generators does not effectively control indoor air pollution, odors, or mold growth on surfaces (Boeniger, 1995; Kissel, 1993; Foarde *et al.*, 1997). Ozone generators can destroy microorganisms and gases, but only at concentrations unsafe for occupied spaces. In addition, ozone from ozone generators can react with indoor surfaces, such as latex paint, or airborne chemicals, including the fragrance compounds from commercial air fresheners, to produce toxic and irritating byproducts such as formaldehyde (Kleno *et al.*, 2001; Wainman *et al.*, 2000; Weschler, 2000; Weschler and Shields, 1999; Moriske *et al.*, 1998; Reiss *et al.*, 1995ab; Weschler *et al.*, 1992). Most importantly, these devices can result in levels of ozone well above health-based standards and guideline levels. For example, a “personal air purifier” tested for its emissions when used according to manufacturer’s directions resulted in ozone levels in the users breathing zone that exceeded various standard levels, including the California ambient air quality standard (Phillips *et al.*, 1999).

Other sources of indoor ozone include other types of electronic air cleaners and office equipment that uses electrostatic processes. Negative ion generators and electrostatic precipitators (ESP) and dry-process copiers, laser printers, and fax machines can generate significant levels of indoor ozone as a by-product (U.S. EPA, 1995; Kissel, 1993; Selway *et al.*, 1980; Allen *et al.*, 1978).

### 2.3 TOXIC AIR CONTAMINANTS AND OTHER INDOOR AIR POLLUTANTS

There are a number of other important indoor pollutants that are somewhat unique in their exposure parameters or health effects. Toxic air contaminants (TACs) are air pollutants “which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health” (HSC Section 39655). Once a compound is identified as a toxic air contaminant, ARB determines the need and appropriate degree of regulation for the compound. Regulations have been implemented to control the release of numerous TACs into outdoor air; however, regulations do not presently exist to control their release into indoor air. Foremost among these pollutants are formaldehyde, chloroform, *p*-dichlorobenzene, benzene, radon, polycyclic aromatic hydrocarbons (PAHs), and environmental tobacco smoke (currently in the identification process). TACs were also identified by the federal government as Hazardous Air Pollutants (HAPs); many are carcinogenic. Semi-volatile pollutants such as pesticides, phthalates, and polybrominated diphenyl ethers can be released indoors, or tracked indoors from outdoors. The health effects, indoor sources, and indoor air concentrations of these pollutants are discussed in this section.

#### 2.3.1 Formaldehyde

Formaldehyde is a pungent smelling gas emitted from numerous indoor sources. These include many building materials (especially pressed wood products), some new carpet assemblies, composite wood furnishings, consumer products, permanent pressed clothing, and combustion sources. Formaldehyde is listed as a TAC and a Proposition 65 substance, based on its carcinogenicity. It is also an upper respiratory tract irritant that produces eye, nose, and throat

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irritation. Indoor formaldehyde concentrations nearly always exceed outdoor levels due to the many indoor sources. Indoor and urban ambient levels typically exceed the OEHHA Chronic Reference Exposure Level (REL) of 2.4 parts per billion (ppb), which is based on irritant effects on the mucous membranes of the upper airways and eyes, and levels sometimes exceed OEHHA's 8-hour REL of 27 ppb designed to protect against the same effects. Nearly all indoor environments also exceed the one-in-a-million cancer risk level.

### 2.3.1.1 *Health Effects of Formaldehyde*

A number of adverse health effects in humans have been associated with formaldehyde exposure. Short-term effects include eye, nose, throat and skin irritation; nausea; headache; and there is limited evidence for exacerbation of asthma. Dermal allergic sensitization may occur following relatively high occupational exposure. People vary substantially in their sensitivity to formaldehyde. For most individuals, effects typically occur at exposure levels between 0.037 and 3 ppm (ARB, 1992; U.S. EPA, 1987b). Sensitive individuals may experience acute symptoms related to irritation at lower concentrations. OEHHA has set the acute REL, based on a one hour exposure, at 94  $\mu\text{g}/\text{m}^3$  (75 ppb) with eye irritation as the toxicological endpoint (OEHHA, 2000a). The OEHHA interim REL, based on an 8-hour exposure, is 27 ppb designed to protect against the same effects.

The ARB identified formaldehyde as a TAC in 1992, based on its carcinogenic potential. In 2004, the International Agency for Research on Cancer (IARC) classified formaldehyde as a human carcinogen due to sufficient evidence for development of nasopharyngeal cancer in humans. IARC also found limited evidence that formaldehyde may cause other respiratory tract cancers, and a possible link with leukemia. The California Proposition 65 No Significant Risk Level for formaldehyde is 40  $\mu\text{g}/\text{day}$  (equivalent to 1.6 ppb, based on inhaling 20  $\text{m}^3/\text{day}$ ). This level represents the daily intake level calculated to result in a cancer risk of one excess case of cancer in 100,000 individuals exposed over a 70-year lifetime (OEHHA, 2004).

### 2.3.1.2 *Sources of Formaldehyde*

Many materials and products emit formaldehyde. However, emissions studies have shown that building materials, particularly composite wood products, are likely the greatest contributors to formaldehyde in indoor air. Kelly *et al.* (1999) reported the highest emission rates for numerous composite wood products manufactured with urea-formaldehyde resin. These products, commonly used in home construction, cabinetry, and furniture, displayed formaldehyde emission rates ranging from 8.6 to 1,580  $\mu\text{g}/\text{m}^2/\text{hr}$ . Over half of the urea-formaldehyde products tested had emission rates between 100 and 200  $\mu\text{g}/\text{m}^2/\text{hr}$ . A covering over the wood such as a paper laminate, melamine laminate, or vinyl coating substantially reduced the emission rates to levels at or below 55  $\mu\text{g}/\text{m}^2/\text{hr}$  for all products tested. Composite wood products designed for outdoor use are made with phenol-formaldehyde resin. When tested, these products emitted 4.1 to 9.2  $\mu\text{g}/\text{m}^2/\text{hr}$  formaldehyde, a substantial reduction from the urea-formaldehyde resin products. Coated products and phenol-formaldehyde resin products are preferred alternatives to urea-resin products.

After numerous lawsuits in the 1970s and 1980s, the composite wood industry developed voluntary emission standards for medium density fiberboard (MDF) and particleboard. A comparison of emission rates from Pickrell *et al.* (1983) and Kelly *et al.* (1999) showed that the emission rates from current composite wood products averaged 49% lower than the emissions in the early 1980s. Industry data provided to ARB by the Composite Panel Association indicate that emissions of particleboard have decreased by 80% in this time frame. In response to an

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ARB survey, members of the composite wood industry responding to the survey (53%) indicated 100% of their particleboard meets the HUD large chamber test concentration of 0.3 ppm (this chamber concentration is not equivalent to the concentration that would be expected in a home). Of the products Kelly *et al.* (1999) tested, all of the bare MDF products and most of the particleboard samples were below the industry limits.

Formaldehyde emissions are greatest when building materials are new, and it takes years to complete the off-gassing. Sexton *et al.* (1986) carefully designed a study to measure indoor formaldehyde levels relative to the age of manufactured homes. Investigators found statistically significant higher formaldehyde concentrations in newer homes than older homes. For example, data collected during the summer indicated the mean concentration for houses less than four years old were 80 ppb, compared to houses older than four years with a mean concentration of 61 ppb. Data from winter sampling reveal mean concentrations of 90 ppb for newer homes and 64 ppb for older homes. Data from the study was further analyzed by the age of the mobile home (Sexton *et al.*, 1989). For homes manufactured from 1980 to pre-1966, the data follow the expected pattern, with indoor values gradually decreasing with increasing age. The highest average concentration was in homes manufactured in 1980. Although homes manufactured in 1981 and 1982 had maximum values greater than those from 1980, the mean concentrations were less. The mean for homes manufactured in 1983 continued this downward trend. Investigators comment that the particle board industry began introducing products with lower formaldehyde emission rates at this time. It is likely that these changes are reflected in the lower formaldehyde levels observed in mobile homes built after 1980.

Composite wood products still release high levels of formaldehyde to the indoor environment for long periods of time—from months to years. Brown (1999), an investigator in Australia, measured formaldehyde emission rates from particleboard, medium density fiberboard, and office furniture over several months. Emission rates measured in the study declined from a value of 300-400  $\mu\text{g}/\text{m}^2/\text{hr}$  for relatively new products to 80-140  $\mu\text{g}/\text{m}^2/\text{hr}$  for products 5 to 10 months old.

In a recent study funded by the California Integrated Waste Management Board (CIWMB) and conducted by DHS (Alevantis, 2003), investigators identified nine products in six categories that emitted formaldehyde at levels high enough to exceed the California Section 01350 guideline level (see Section 4.3.3.2 of this report). When modeling (for use in a state office) was conducted independently on emissions from acoustical ceiling panels, a carpet, medium density fiberboard, gypsum board, resilient flooring (non-rubber based), and thermal insulation, room concentrations were estimated to exceed 16.5  $\mu\text{g}/\text{m}^3$  (13.5 ppb), the upper bound allowed for formaldehyde contribution from a single product under Section 01350 guidelines. For thermal insulation, products exceeding the upper limit included a standard product and a product marketed as formaldehyde-free. Only one of the ten standard resilient flooring samples exceeded the limit, while six of the samples had undetectable levels of formaldehyde. The nine products with elevated emissions accounted for 11% of the samples tested in this study. Formaldehyde was detected in 34% of the total samples tested.

Since the creation of the Section 01350 emission guideline for building materials, many manufacturers have met the requirement for a variety of products. A list of these compliant materials is available at [http://www.chps.net/manual/lem\\_table.htm](http://www.chps.net/manual/lem_table.htm).

Recent studies in other states provide additional information on source contributions of indoor formaldehyde. In a manufactured house produced in Florida, Hodgson *et al.* (2002) determined that the greatest contributors to indoor formaldehyde levels were a particleboard cabinet case and passage doors, each contributing about 33% of the total house formaldehyde

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concentration. The house, which was a furnished sales model, had an indoor formaldehyde concentration of 77 ppb. The combined emission rate for all wood products in the house was approximately 10 mg/hr. The other 33% of formaldehyde was contributed by cabinet stiles, countertops, subflooring, and other sources. The authors recommended several techniques for reducing indoor residential formaldehyde levels.

- Use vinyl coated or alternative passage doors.
- Use fully coated particleboard in cabinet cases.
- Use frameless cabinets.
- Apply a laminate backing to the underside of particleboard countertops.

Floor finishing materials such as new carpet assembly components and vinyl flooring may also emit formaldehyde. In a study funded by the ARB, Hodgson (1999) measured the formaldehyde emissions of several flooring products. Results for formaldehyde emissions at 24 hours showed the following.

- Formaldehyde chamber concentrations were generally below the limit of detection of 1  $\mu\text{g}/\text{m}^3$  for carpet.
- One carpet cushion had an emission rate of 8  $\mu\text{g}/\text{m}^2/\text{hr}$ .
- Seam tape applied to carpet had an emission rate of 5  $\mu\text{g}/\text{m}^2/\text{hr}$ .
- Emissions from five different sheet vinyl flooring samples were less than or equal to 4  $\mu\text{g}/\text{m}^2/\text{hr}$ .
- Adhesives applied to sheet flooring and cove base had emission rates ranging from 72 to 258  $\mu\text{g}/\text{m}^2/\text{hr}$ .
- When the vinyl flooring or coving was placed on top of the adhesive, the surface product served as a relatively effective barrier, causing a drop in emissions.

Paint is another building material known to emit formaldehyde. Hodgson (1999) measured formaldehyde emissions from 10 different paints widely used in California. As with most wet products, concentrations peaked initially, then declined over several hours. At 96 hours, five paints had emission rates greater than 10  $\mu\text{g}/\text{m}^2/\text{hr}$ . To address the desire for lower-emitting products, many paint manufacturers are formulating low-VOC paints. These paints have substantially lower total VOC emissions than traditional paint; however, they may emit formaldehyde. Two of the paints in the study were identified as “non-VOC”, however their formaldehyde emissions rates at 48 hours were 43 and 12  $\mu\text{g}/\text{m}^2/\text{hr}$ . Chang *et al.* (1999) also evaluated emissions from low-VOC paints. In small chamber tests, two of four low-VOC paints emitted formaldehyde with peak concentrations at 3.15  $\text{mg}/\text{m}^3$  and 5.53  $\text{mg}/\text{m}^3$ .

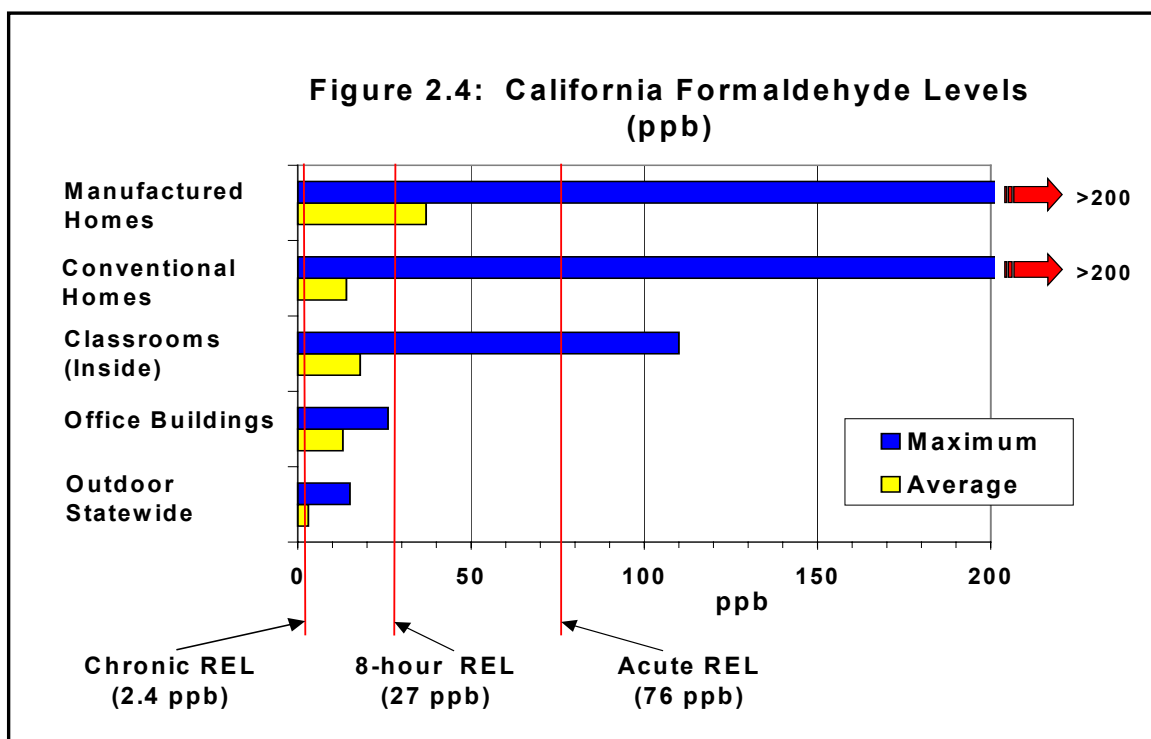
### 2.3.1.3 Indoor Formaldehyde Concentrations

Indoor levels of formaldehyde can reach high levels in some indoor locations despite changes in the manufacture of pressed wood products and changes in the construction of manufactured housing. Current estimated average and maximum indoor formaldehyde concentrations are shown in Figure 2.4 for different California environments. Figure 2.4 illustrates that concentrations in some homes and schools exceed OEHHA’s interim 8-hour REL of 27 ppb, established to identify levels above which sensitive individuals could experience acute eye, nose, and lung irritation. The figure also shows the relative levels of formaldehyde in different environments, indicating that manufactured homes are of greatest concern, while conventional homes and classrooms are also of concern.

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Figure 2.4 also illustrates that levels in virtually all indoor environments exceed OEHHA's chronic REL (2.4 ppb) for irritant effects and OEHHA's one-in-a-million excess lifetime cancer risk level (0.13 ppb) for formaldehyde. It is generally not feasible to achieve levels below these guideline levels because outdoor levels average about 3-5 ppb.

Data for Figure 2.4 have been compiled from several sources. Due to the lack of more recent field studies that measured indoor formaldehyde levels, adjustments have been made to the results from older studies. Manufactured home levels are from a large, older study of manufactured homes (Sexton *et al.*, 1985), with levels adjusted downward based on the



average reduction in emissions from manufactured wood products since 1983 (Kelly *et al.*, 1999). The conventional home data were obtained by combining results from a 1996 study of southern Californian homes (Avol *et al.*, 1996) and the National Human Exposure Assessment Survey (NHEXAS) exposure study in Arizona (Gordon *et al.*, 1999). The maximum values estimated for current California manufactured and conventional homes are similar, between 220-240 ppb, although measured values have been obtained well above these levels in some studies. The classroom data are taken from the California Portable Classrooms Study (Whitmore *et al.*, 2003), using both Phase I and Phase II data. Concentrations in office buildings are from the U.S. BASE study (U.S. EPA, 2003a; Girman *et al.*, 1999) of about 100 medium and large office buildings throughout the U.S., including buildings from California. The sources and derivations of the averages and maxima shown in Figure 2.4 are discussed in Appendix III.

New homes built with standard construction products are likely to have unhealthy levels of formaldehyde. Hodgson *et al.* (2000) measured formaldehyde concentrations inside homes two months old, then continued measurements for a seven and one-half month period. The geometric mean concentration of formaldehyde in four manufactured homes was 34 ppb, and in seven site-built homes was 36 ppb. Both were substantially greater than the outdoor formaldehyde level of 6 ppb, and above health-based guidelines. The formaldehyde emission

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rates were fairly consistent over the seven to eight month sampling time, indicating that the off gassing of formaldehyde emissions from building materials in new homes extends for a long period of time. The author commented that these levels are approximately 50% lower than formaldehyde concentrations measured during the 1980s due to use of less plywood paneling in manufactured homes and reduced emission rates from composite wood products relative to 20 years ago. Emission rates were calculated for these homes and found to be 45  $\mu\text{g}/\text{m}^2/\text{hr}$  for the manufactured homes and 31  $\mu\text{g}/\text{m}^2/\text{hr}$  for the site-built homes.

Older housing stock has greatly reduced formaldehyde off-gassing rates relative to new housing stock. As mentioned in Section 2.3.1.2, longitudinal formaldehyde concentration studies extending over years do not exist and emission rates are generally not derived for older houses. However, the body of data on home formaldehyde concentrations indicates newer homes have higher levels than older homes (Hodgson *et al.* 2000; Gordon *et al.*, 1999). Therefore, new homes, or homes with recent remodeling are of most concern relative to formaldehyde exposure.

In the most recent large-scale study of formaldehyde levels in homes (NHEXAS), conducted in Arizona, 25% of the homes sampled had formaldehyde levels above the 8-hour REL of 27 ppb. This is of concern because people are typically in their homes for longer than 8 hours a day and the study was designed to be representative of the general population. The formaldehyde limit of detection was fairly high at 10 ppb, so that formaldehyde was detectable in only 69% of the indoor air samples (131/189) with a median concentration of 21  $\mu\text{g}/\text{m}^3$  (17 ppb). The 75<sup>th</sup> and 90<sup>th</sup> percentiles in residences were 34  $\mu\text{g}/\text{m}^3$  (28 ppb) and 46  $\mu\text{g}/\text{m}^3$  (37 ppb), respectively, with a maximum value at 408  $\mu\text{g}/\text{m}^3$  (332 ppb). These investigators also commented that mean values are lower than those reported in the 1980s (Gordon *et al.*, 1999).

Although not evident in Figure 2.4, the results from Phase II of the California Portable Classroom Study (PCS) indicated that formaldehyde levels in at least 4% of California classrooms exceed OEHHA's interim 8-hour REL of 27 ppb (Whitmore *et al.*, 2003), the level at which an 8-hour exposure might result in irritant effects. This is equivalent to about 10,720 classrooms, or at least 214,400 children (assuming 20 children per classroom...usually there are more) exposed to formaldehyde levels that could potentially result in irritant effects in sensitive individuals. Average and peak levels in the classrooms were somewhat higher than those measured in the U.S. EPA BASE study of public and commercial buildings, indicating that, during the day, school children may experience greater exposures than most adults. Data analyses from the PCS indicate that several factors were associated with indoor formaldehyde levels in classrooms including the presence of plywood and particleboard; vinyl tackboard; bookcases and cabinets made of pressed wood; increased temperature and humidity; and classroom age (higher levels in newer classrooms) (Whitmore *et al.*, 2003).

### 2.3.2 Volatile Organic Chemicals

Volatile organic chemicals (VOCs) is a generic term for thousands of compounds with widely varying physical, chemical, and toxicological properties. VOCs include a variety of chemicals such as alkanes, alcohols, esters, ethers, and aromatic compounds. A number of specific VOCs are widely found in indoor environments and are known to have adverse toxicological properties. Potential indoor sources of VOCs include building and furnishing materials such as carpet, paint, and vinyl flooring; consumer products such as air fresheners, adhesives, and cleaning agents; water treated with chlorine; dry-cleaned clothing; environmental tobacco smoke; plastic products, computers, and others.

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Many of the 'traditional' VOCs reported in indoor air have been studied simultaneously because, due to their chemical properties, they are collected via the same medium and analyzed through the same processes. Scientific study has only touched the 'tip of the iceberg' in understanding all VOCs in indoor air. Complex compounds such as fragrances are widely used, minimally regulated, and an area of emerging concern for environmental impacts (Bridges, 2002). Many semi-volatile compounds that can be present in the gaseous phase, or adsorbed to particles have not been extensively studied (Rudel *et al.*, 2003). Because the link between indoor air pollutants and health impacts is so complex, researchers are investigating the reaction of VOCs with oxidants present in indoor air and are discovering new areas for future study (Carslaw, 2003; Weschler, 2004; Wolkoff and Nielsen, 2001; Wolkoff *et al.*, 2000).

Some VOCs have been identified as California TACs or federal HAPs due to their cancer-causing potential. Some VOCs also cause eye, nose, and throat irritation, and neurological effects such as headache. Indoor and personal exposure levels of these VOCs are typically higher than concurrent outdoor levels, and may exceed acceptable cancer risk levels and other health-based guideline levels. Ambient VOC levels are generally considered 'background' levels for indoor pollutants due to infiltration of outdoor air, thus any additional pollutant released indoors results in a concentration greater than outdoors.

### 2.3.2.1 Health Effects of VOCs

The ability of organic chemicals to cause health effects varies greatly from those that are highly toxic, to those with no known health effect. Some of the VOCs found in indoor air in California have been identified as TACs based on their carcinogenic potential. Accordingly, there is no level of exposure to these chemicals that is known to be absolutely safe. However, health effects are determined not only by the specific toxicology of the air pollutant but also by the extent of exposure and the absorbed dose. The higher the exposure and dose, the higher the risk of adverse health effects. More information on dose-response can be found on the OEHHA website ([www.oehha.ca.gov](http://www.oehha.ca.gov)). Some of the more common carcinogenic indoor VOCs were listed earlier in Table 2.4. The cancer risk posed by some of the most prevalent VOCs was discussed above in Section 2.1.2, and a detailed discussion of those risks is provided in Appendix II.

Many indoor VOCs also can irritate the eyes, nose, and throat (OEHHA 2000a; OEHHA 2003a). They are emitted from a wide range of sources and have a variety of impacts on occupant health and comfort (Hodgson *et al.*, 1994; Wolkoff, 1995; Molhave, 1991b). Hodgson and Levin (2003a) compared indoor VOC concentrations to odor thresholds, sensory irritation levels, and noncancer chronic health guidelines. "The methodology demonstrated that only a small number of the more than 100 reported indoor VOCs exceeded levels likely to be of concern with respect to the endpoints considered. The results indicated carboxylic acids and less volatile aldehydes and aromatic hydrocarbons are most likely to be perceived by olfaction and that the probability of detection is higher in residences than in offices. Although more consideration of the underlying toxicological data is needed, the results suggested only a few commonly measured VOCs, considered singly, are likely to produce serious irreversible health effects not associated with cancer". The authors conclude, "For a few compounds, such as acrolein and formaldehyde, the evidence based on sensory irritation and chronic toxicity is sufficient to warrant efforts to reduce and otherwise control the sources of these compounds in buildings."

At higher concentrations (usually not encountered in homes or offices, but sometimes found in occupational settings), some VOCs can impact the nervous system, causing acute effects such as nausea, tremors, drowsiness, dizziness, and headache (OEHHA 2000a; OEHHA 2003a). Such VOCs include, most notably, aromatic hydrocarbons (such as benzene), chlorinated



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chemicals (such as perchloroethylene), and some pesticides. While concentrations that can trigger neurotoxic effects are not normally experienced in homes and offices, special circumstances can lead to elevated exposures in some specialized non-industrial workplaces. The nature of activities and products used in medical offices, hospitals, beauty salons, high production copy shops, and other non-industrial workplaces can lead to unusually high concentrations of some pollutants. Business parks that merge industrial and non-industrial businesses may also create unique situations leading to exposure to elevated levels of these pollutants.

Exposure of pregnant women to organic solvents may affect the neurodevelopmental outcome of their children. After controlling for potential confounding related to maternal IQ and maternal education, Laslo-Baker *et al.* (2004) found that children exposed in utero to organic solvents obtained lower scores on subtests of intellect, language, motor, and neurobehavioral functioning. Many of the occupations were non-industrial and included painter, hair stylist, salon receptionist, and science teacher. Exposure levels were not reported.

### 2.3.2.2 Sources and Emissions of VOCs

Studies have shown that many indoor sources emit VOCs, that some indoor sources emit substantial amounts of VOCs, and that groups of related VOCs often have common sources. Exposure to VOCs is influenced by people's activities and their proximity to sources of pollutants. Exposure studies indicate the greatest exposures generally result from close contact with specific sources, such as cleaning products. Manufacturers strive to produce products that meet consumers' needs and can be used safely; however, VOCs are often required in the manufacture of products to impart desired properties for a given application. This results in trade-offs: proper use of some cleaning products, for example, can remove biological contaminants and some allergens and asthma triggers in the indoor environment, yet occupants sensitive to the odor and irritant effects of the VOC components may be affected.

#### Chlorinated Solvents

Chlorinated hydrocarbons, a large group of VOCs with solvent properties, contain one or more chlorine atoms. Chemicals in this group have diverse sources. Levels of perchloroethylene, identified as a California TAC due to its carcinogenic potential, can increase when dry-cleaned clothes are brought into a house. Levels in a home containing recently dry-cleaned clothes can be 100 to 150 times greater than outdoor levels of perchloroethylene (Wallace, 2001). Levels of methylene chloride, another chlorinated hydrocarbon and TAC, have been greatly reduced in consumer products; however, it is still common in paint strippers with a label warning to use adequate ventilation. Short-term exposures can be significant for individuals who use paint strippers (Wallace, 1991).

*Para*-dichlorobenzene (*p*-DCB), another chlorinated solvent, has been used as an "air freshener," although it is listed as a TAC due to its potential carcinogenicity. Data from the U.S. EPA's TEAM studies indicated that, at the time the study was conducted, about one-third of homes used products containing *p*-DCB (Wallace, 1991). In another study, the indoor air concentration of *p*-DCB increased from 1 µg/m<sup>3</sup> to more than 500 µg/m<sup>3</sup> the day after a toilet bowl cleaner was introduced into a home (Wallace, 2001). In the NHEXAS study, the two highest *p*-DCB levels measured in Arizona homes were 3949 and 4400 µg/m<sup>3</sup>, presumably from the introduction of household consumer products (Gordon *et al.*, 1999). These examples illustrate the extremely high concentrations of chlorinated chemicals that can occur in residences.

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Chlorine is intentionally added to domestic water for public health purposes. However, chlorine and organic matter in the water react to produce chloroform, another compound listed as a California TAC. Exposure to chloroform and other trihalomethanes occurs from drinking water, taking showers and baths, and operating washing machines (Wallace, 2001). In one study, investigators measured chloroform concentrations while individuals actually took showers in residential shower stalls. Average concentrations during and immediately after the shower ranged from 67  $\mu\text{g}/\text{m}^3$  to 265  $\mu\text{g}/\text{m}^3$ . The chronic REL for chloroform is 300  $\mu\text{g}/\text{m}^3$  (OEHHA, 2003a). Air concentrations of trihalomethanes were about three times higher on average for a typical shower compared to a bath. Higher concentrations were observed with hotter water (Kerger *et al.*, 2000). Chloroform can also be produced during the use of bleach and other cleaning agents that contain chlorine. Investigators predicted that 5.3 mg to 9.8 mg of chloroform can be released to indoor air during a ten-minute wash cycle when a laundry bleach containing sodium hypochlorite is used in a residential washing machine (Shepherd *et al.*, 1996). If this amount of chloroform is released into a 10 foot by 10 foot room (22.7  $\text{m}^3$ ), the chloroform concentration would be 230 to 430  $\mu\text{g}/\text{m}^3$ , the high end of which would exceed the chronic REL.

### Benzene and Other Aromatic Hydrocarbons

Benzene, styrene, toluene, and xylene are aromatic hydrocarbons containing a hexagonal ring structure. In general, the major sources of these chemicals in the indoor environment are environmental tobacco smoke, motor vehicle exhaust, and evaporative emissions from vehicles in attached garages. To a lesser extent, consumer products such as solvents, adhesives, glues, and paint emit some of these VOCs, especially toluene (Wallace *et al.*, 1988; Akland and Whitaker, 2000; Guo *et al.*, 1999). The overwhelming source of benzene exposure for smokers is mainstream cigarette smoke (Wallace, 1996b); nonsmokers living with smokers may experience a substantial benzene exposure due to ETS as well (Wallace, 2001). For nonsmokers with infrequent exposure to ETS, most benzene exposure comes from auto exhaust, especially while traveling on busy roadways (Rodes *et al.*, 1998), gasoline vapor emissions during fueling at gas stations, and in houses with an attached garage (Wallace, 1996b).

Some of these VOCs are also emitted from office copy machines and printers (Lee *et al.*, 2001). Several investigators have studied the emission of aromatic hydrocarbons from office machines. Numerous VOCs, including benzene, ethylbenzene, *o,m,p*-xylene, and styrene were emitted from a dry-process photocopier studied by Brown (1999). The author noted a 40% increase in VOC emission rates for double-sided operation versus single-sided operation. Leovic *et al.* (1996) measured emissions from 4 dry-process photocopier machines. The compounds with the highest emission rates overall were ethylbenzene (highest emission at 28,000  $\mu\text{g}/\text{hour}$ ), *o,m,p*-xylene, 2-ethyl-1-hexanol, and styrene (lowest emitter of these compounds at 12,000  $\mu\text{g}/\text{hour}$ ). The relative amounts of individual compounds varied with the machine. Other investigators (Wolkoff *et al.*, 1993) measured emissions from toners and processed paper from office copiers and printers. They also report substantial variation in emissions between machines. Aromatic compounds such as toluene, xylenes, ethyl and propyl benzene, and styrene dominated the emissions. The authors concluded "a realistic estimate (assuming first order decay) of handling 200 freshly processed copies in a 17  $\text{m}^3$  office room, 0.25/hour air exchange rate, and an emission of 6  $\mu\text{g}/\text{m}^2\text{hr}$  could reach a styrene concentration, assuming complete mixing, of 12  $\mu\text{g}/\text{m}^3$  from the processed paper." This concentration is well below the chronic REL of 900  $\mu\text{g}/\text{m}^3$ , however results indicate total emissions including ozone and particles can have a significant impact on indoor air quality (Wolkoff *et al.*, 1993; Leovic *et al.*, 1996).

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In an unpublished study of over 100 products sold in California stores, Akland and Whitaker (2000) detected toluene most frequently in auto care products, glues, and personal care products, such as in fingernail polishes. Estimated maximum air concentrations and daily doses were high, up to 4000 µg/day. In the Arizona NHEXAS study, investigators measured toluene levels at non-smokers' homes and found an average level of 24 µg/m<sup>3</sup> in homes with an attached garage (n=40), but only 5 µg/m<sup>3</sup> in homes without an attached garage (n=9) (Gordon *et al.*, 1999). These concentrations are below the chronic inhalation REL for toluene of 300 µg/m<sup>3</sup> (OEHHA, 2003a).

### VOCs from Building Materials

The pollutants discussed above are associated with one or a few predominant sources. But, many sources, such as building materials emit numerous VOCs. A number of studies have been conducted to examine VOC emissions from a variety of building materials. Most recently, a building material emissions testing study funded by CIWMB reported that building materials emit a number of VOCs that were identified as chemicals of concern (Alevantis, 2003). Several products in each of the categories identified below exceeded the Section 01350 guideline levels as follows:

- Carpet exceeded the emission rate limits for naphthalene and acetaldehyde.
- Fiberboard and particleboard exceeded the limits for acetaldehyde.
- Resilient flooring products exceeded the limit for acetaldehyde, naphthalene, and phenol.

As was mentioned in the section on formaldehyde, products meeting Section 01350 emission guidelines are listed at [http://www.chps.net/manual/lem\\_table.htm](http://www.chps.net/manual/lem_table.htm).

In a study funded by ARB, Hodgson (1999) identified 17 toxic air contaminants in the emissions from new carpet assembly, vinyl flooring, and latex paint. These TACs are routinely emitted to the indoor environment, particularly in new or recently remodeled homes and offices. Table 2.6 contains a list of TACs identified by Hodgson in building material emissions. In addition to TACs, all of the bonded urethane carpet cushions emitted butylated hydroxytoluene (BHT), an irritant, and all carpet samples emitted 4-phenylcyclohexene (4-PC), the compound largely responsible for new carpet odor. The investigator tested a limited number of products on the market at the time. The study conducted by Alevantis (2003) contains more recent emissions data, reflective of current manufacturing processes. It also compares emissions to health-based guidelines.

**Table 2.6. Toxic Air Contaminants Emitted From Building and Finishing Materials**

<b>Carpet and Cushion</b>	<b>Paint</b>	<b>Vinyl flooring</b>
Toluene	<i>m,p</i> -Xylene	Toluene
<i>m,p</i> -Xylene	Ethylene glycol	<i>m,p</i> -Xylene
<i>o</i> -Xylene	2-(2-Butoxyethoxy)ethanol	<i>o</i> -Xylene
Styrene	Formaldehyde	Styrene
Ethylene glycol	Acetaldehyde	1,2,4-Trimethyl benzene
Formaldehyde		Naphthalene
Acetaldehyde		Acetophenone
Acetophenone		Phenol
2-(2-Butoxy)ethanol		Formaldehyde
Ethylbenzene		Acetaldehyde
Tetrachloroethane		
Naphthalene		
Phenol		

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Emissions from polymeric building materials can be attributed to several factors (Yu and Crump, 1998).

- Solvent residues from the manufacturing process.
- Unreacted monomers trapped in the product structure.
- Secondary products from reactions of monomers.
- Plasticizers used in production.

Measurement of VOCs in new homes is another method of determining the variety of compounds emitted from building materials. Hodgson *et al.* (2000) identified  $\alpha$ -pinene, formaldehyde, hexanal, and acetic acid as the predominant compounds measured in 11 new homes. For manufactured houses and site-built houses, the geometric mean total volatile organic chemicals (TVOC) emission rate was 1.7 mg/m<sup>2</sup>-h, and 2.1 mg/m<sup>2</sup>-h, respectively. Individual compounds with the highest emission rates (greater than 50  $\mu$ g/m<sup>2</sup>-h) include  $\alpha$ -pinene, ethylene glycol, hexanal, acetic acid,  $\beta$ -pinene, and 2,2,4-trimethyl-1,3-pentanediol monoisobutyrate (TMPD-MIB).

Paint not only emits numerous TACs, but emissions can continue for extended periods of time. Sparks *et al.* (1999) estimated that less than 50% of the VOCs in latex paint are emitted in the first year. Compounds studied include ethylene glycol, propylene glycol, Texanol®, and butoxyethoxyethanol. Alkyd paints contain substantially more VOCs than latex paints due to the use of mineral spirits as the solvent. Compounds emitted from alkyd paints include alkanes such as decane, nonane, octane, undecane, and aromatic compounds such as xylenes, toluene, and ethylbenzene (Guo *et al.*, 1999).

It is becoming increasingly apparent that VOCs emitted into indoor air can undergo chemical reactions. Morrison and Nazaroff (2002) studied the reaction of ozone with VOCs emitted by carpet to produce aldehydes. Of particular interest was the production of 2-nonenal, a compound with a low odor threshold. Investigators estimated that 2-nonenal could be generated from the carpet emissions at rates leading to odor detection for longer than three years.

### VOC Emissions from Consumer Products

Consumer products such as cleaning products, personal care products, art supplies, and hobby supplies can release pollutants to the indoor environment and cause high personal exposures to pollutants. Wallace *et al.* (1989) measured the impact of activities and product use on personal exposure to VOCs, several of which are carcinogens. Breath levels of VOCs were often significantly correlated with previous personal exposures. Use of consumer products was associated with a variety of increased exposures as follows: use of deodorizers (*p*-dichlorobenzene), washing clothes and dishes (chloroform), visiting a dry cleaners (1,1,1-trichloroethane, tetrachloroethylene), smoking (benzene, styrene), cleaning a car engine (xylenes, ethylbenzene, tetrachloroethylene), and painting and using paint remover (n-decane, n-undecane).

During the late 1980s and early 1990s, the U.S. EPA conducted an indoor air quality research program to develop test protocols, determine emission factors of products, and develop emission models (Tichenor, 1989). During this time much was learned about emission profiles, sink effects, and the VOCs emitted by various consumer products. A list of organic compounds was identified in products such as paint, stain, adhesive, furniture polish, and caulk (Tichenor, 1989; Tichenor and Mason, 1988). These early studies were instrumental in understanding the role consumer products play in indoor air quality. However, changes in product formulations make newer studies more appropriate for understanding current indoor air quality impacts.

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In an unpublished study, Akland and Whitaker (2000) found six products that they estimated would exceed the Proposition 65 no-significant-risk level for formaldehyde, including a nail finish, make-up, floor-cleaning spray, formica laminate, foaming tire care, and sheet flooring adhesive. Two products exceeded the no-significant-risk level for benzene, one product exceeded the level for acetaldehyde, and another product exceeded the level for tetrachloroethylene. The authors state "Comparison with the California levels would be reasonable only to the extent that the emission testing conditions represent typical indoor conditions, and that the product emissions actually resulted in exposure to the person for a 24-hour period". Products were applied to a glass substrate, so emissions may be different than would be expected in a typical situation.

Cleaning products have been studied to assess their impact on personal exposure in indoor environments. Zhu *et al.* (2001) tested 13 cleaning products to assess the potential for human exposure to three glycol ethers. 2-Butoxyethanol (BE) was present in the head space samples of seven products, of which five were household cleaning agents. BE concentration in the products ranged from 0.50 to 3.72%. 2-Methoxyethanol (ME), 2-ethoxyethanol (EE) were not detected; both of these are more toxic than BE. Investigators calculated average daily inhalation exposure levels for an individual cleaning with these products at home. Calculations were based on product use scenarios developed by U.S. EPA and a "standard room". For two all-purpose spray cleaners the average daily inhalation exposure for 2-butoxyethanol was 0.075 and 0.186 mg/kg body weight/day, and for two spray glass cleaners it was 0.004 and 0.006 mg/kg body weight/day. The high-end exposure of 0.186 mg/kg body wt/day for a 71 kg person would be 13.2 mg/day. This is below the U.S. EPA's inhalation reference concentration (RfC) for BE of 13 mg/m<sup>3</sup>, the daily inhalation exposure that is likely to be associated with an appreciable risk of deleterious effects during a lifetime (U.S. EPA, 1999c). (To compare product emissions to the RfC, multiply the RfC by 20 m<sup>3</sup>/day [daily breathing rate] to yield 260 mg/day, which is notably greater than the concentrations measured during the cleaning protocols.)

Other investigators also have studied emissions from consumer products and personal care products. Cooper *et al.* (1995) identified ethanol,  $\alpha$ -pinene, camphene,  $\beta$ -pinene, diethylene glycol monoethyl ether, benzaldehyde and others as components of fragrance in two colognes, a perfume, a soap, and an air freshener. Toxicological data indicate the compounds may be irritants, mutagenic, teratogenic, or carcinogenic; however, risk of these health effects may be low given typical use of these products.

The impact of human occupancy and activities on VOC concentrations was documented in a study conducted by Shields *et al.* (1996). Investigators measured VOC concentrations indoors and outdoors at 70 offices having ranges in occupant density. The results showed that VOC concentrations were associated with the density of occupancy and ventilation rate. The authors were able to identify six compounds associated with occupant density: limonene used in cleaning products and air fresheners; tetrachloroethylene from dry-cleaned clothes; n-dodecane through n-hexadecane, probably from cosmetics, hand lotions, and shaving creams; and octa- and deca-methylcycllopentasiloxanes, which are associated with underarm deodorants and antiperspirants. It is important to note that the compounds associated with personal care products were present at low concentrations and they are not TACs or on the Proposition 65 list. The database generated by the study can be used to identify atypical compounds or concentrations in office buildings.

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### 2.3.2.3 Indoor Concentrations of VOCs

Several major studies of concentrations of selected toxic pollutants in California residences have been conducted under the sponsorship of the U.S. EPA and/or ARB. Studies of VOCs were conducted over different seasons in three different years (1984, 1987, and 1990) in a total of nearly 500 California homes (Wallace, 1991; Wallace *et al.*, 1988; Sheldon *et al.*, 1992a). Analyses of the results across these and other studies indicate that indoor VOC concentrations are typically two to five times higher, and sometimes many times higher, than outdoor air (Wallace, 1991; Wallace *et al.*, 1988; Sheldon *et al.*, 1992a; Ott and Roberts, 1998). Results also indicate that personal (actual) exposure levels are generally higher than either indoor or outdoor residential levels. These results were consistent across different seasons and different geographic locations.

#### California Indoor VOC Studies

The average and 90<sup>th</sup> percentile personal, indoor, and outdoor concentrations of several VOCs measured in a northern California study are presented in Table 2.7 (Sheldon *et al.*, 1992a). Personal levels are usually higher than indoor and outdoor levels due to people's frequent use of, or proximity to, sources such as consumer products as they go about their daily activities. Table 2.7 includes a risk level for chemicals listed on the Prop 65 list. At the risk level presented, one excess cancer case per 100,000 individuals may be expected over a 70-year lifetime. The table indicates that a substantial portion of the population could be exposed to levels of *para*-dichlorobenzene, benzene, and perchloroethylene that pose an unacceptable excess cancer risk.

**Table 2.7. Personal, Indoor, and Outdoor VOC Concentrations  
From a Northern California Study<sup>1</sup>  
( $\mu\text{g}/\text{m}^3$ ; means and 90<sup>th</sup> percentile)**

Compound	Cancer Risk Level <sup>2</sup>	Personal Concentration		Indoor Concentration		Outdoor Concentration	
		mean	90	mean	90	mean	90
<i>para</i> -dichlorobenzene	1.0	21	88	18	36	0.30	0.94
Benzene	0.65	5	8.9	4.7	8.3	1.2	1.9
Perchloroethylene	0.7	1.6	3.0	1.1	2.3	0.53	0.59
Trichloroethylene	4.0	2.3	3.4	0.68	2.0	NQ <sup>3</sup>	NQ
Styrene	Not listed	2.4	3.3	2.8	3.9	0.24	0.70
1,1,1-Trichloroethane	Not listed	22	36	6.5	11	1.5	1.9
<i>m,p</i> -xylene	Not listed	9.3	18	6.3	13	1.8	2.9

1. From Sheldon *et al.*, 1992a.
2. Air concentrations that should not be exceeded to meet Proposition 65. For Prop 65, the "no significant risk" level is defined as 1 excess case of cancer per 100,000 individuals exposed over a 70-year lifetime.
3. NQ = not quantifiable: below the method quantifiable limit.

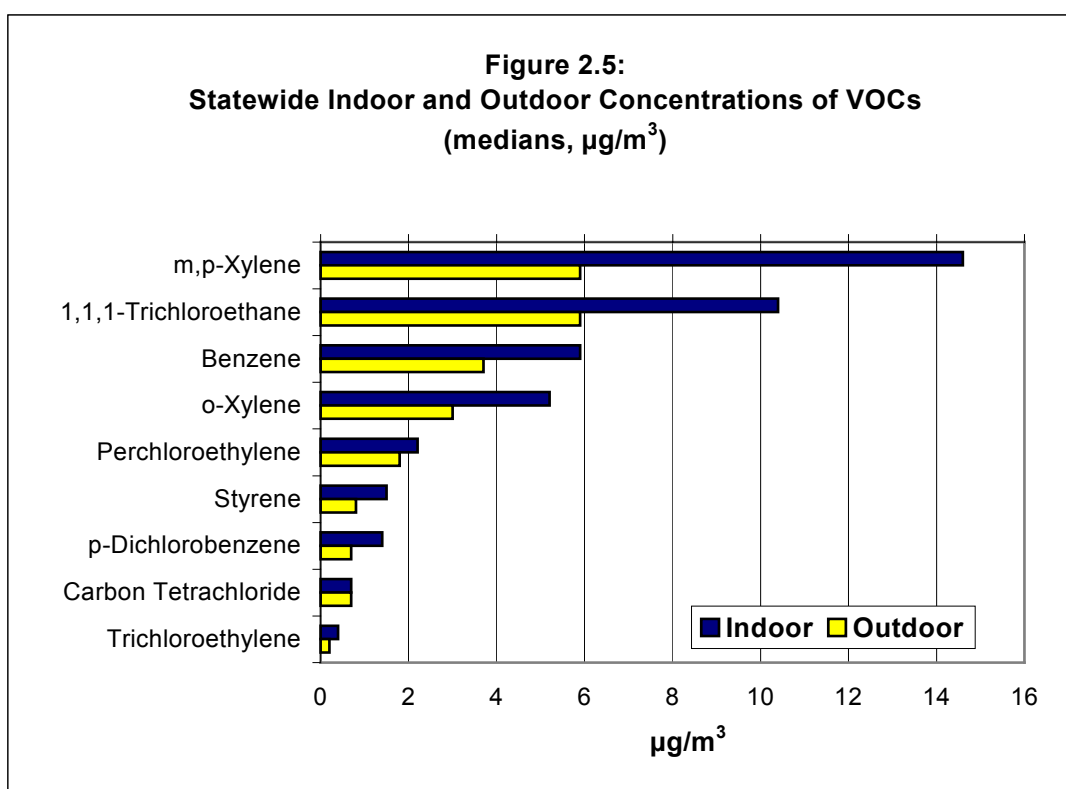
Another example of California VOC indoor and outdoor concentrations is presented in Figure 2.5. Figure 2.5 summarizes the median levels (levels at which half of the homes are higher and half lower) of indoor and outdoor concentrations of selected VOCs using combined data from one of the California VOC TEAM studies (February and July, 1987 in Los Angeles; Pellizzari *et al.*, 1989) and a study conducted in Woodland, California that was similar to the TEAM studies

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(Sheldon *et al.*, 1992a). Figure 2.5 reflects the presence of indoor sources for all chemicals shown except for carbon tetrachloride, for which there are no longer indoor sources because of product restrictions imposed at the national level (CPSC, 1987b). Benzene concentrations measured in Texas homes during 1993 (Mukerjee *et al.*, 1997a,b) were comparable to levels measured in the California studies. It is important to note that the indoor concentrations presented in Figure 2.5 are less than the corresponding chronic REL for each given pollutant by a factor of six, or greater.

### Trends

Some spatial and temporal trends in VOC levels (not illustrated in the figure) also have been noted: indoor concentrations in southern California appear to be somewhat higher than indoor levels in northern California, and indoor levels in winter tend to be higher than indoor levels in summer (Jenkins *et al.*, 1992b).



Some additional trends found in indoor air VOC studies were identified by Brown *et al.* (1994) in their review of studies conducted between 1978 and 1990. The review encompasses measurements from residences, offices, schools and hospitals. Significant findings include:

- For all compounds, indoor concentrations were greater than outdoor concentrations by a factor of two to 73, indicating indoor sources were present. Mean VOC and TVOC concentrations were generally greater in established residences than established public buildings.
- New buildings had considerably higher TVOC concentrations than established buildings. For example, the weighted-average geometric mean TVOC concentration from 33 new residences was  $4,500 \mu\text{g}/\text{m}^3$ , whereas it was  $1,130 \mu\text{g}/\text{m}^3$  from 1,081 established buildings.

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- VOCs specific to new buildings included 2-ethoxyethylacetate, n-butanol,  $\alpha$ -pinene, undecane, dodecane, tridecane, tetradecane, and 2-propanol.

### Apparent Changes in Concentrations Over Time

Levels of some VOCs in California homes, such as benzene, have ~~most likely~~ decreased since these studies were conducted, due to changes in some building materials and consumer products, and the reduced smoking rates in homes. Some studies from other locations appear to support this, although others indicate continued levels of concern.

In a review of 12 studies conducted in North America. Hodgson and Levin (2003b) analyzed residential VOC concentration data collected from 1990 through 2003. They hypothesized that the 1990 Clean Air Act Amendments impacted indoor VOC concentrations through reduced infiltration of ambient pollutants and through industry changes in consumer products and other materials used indoors to use less toxic compounds. Hodgson and Levin found 1,1,1-trichloroethane concentrations to be more than three times lower than the TEAM study value. Benzene, 1,2-dichloroethane and tetrachloroethene were approximately one-half an order of magnitude lower in the current review. The authors noted that the data were limited in a number of aspects. They also highlight that “despite these trends, indoor exposures to most common VOCs undoubtedly still dominate human exposures to these compounds”. In another study, VOC concentrations for five out of six VOCs measured recently in Baltimore, Maryland residences (Payne-Sturges *et al.*, 2004) were somewhat lower than levels shown in Figure 2.5. However, there were differences in sample collection methods (passive samplers in Baltimore, active samplers in California) and undoubtedly differences in housing characteristics based on region. The focus of the Baltimore study was to estimate cancer risk. Although concentrations are lower than measurements taken from studies conducted during the 1980s, the authors estimate a cumulative indoor cancer risk of 120 per million based on the median indoor concentrations.

Recent studies in other states continue to show higher levels indoors than outdoors, and indoor levels above health risk guidelines. For example, results from the recent Arizona National Human Exposure Assessment Survey (NHEXAS) indicate that indoor levels of pollutants continue to be greater than outdoor levels, and some are comparable to levels measured in the initial TEAM studies (Gordon *et al.*, 1999). In another recent study conducted in Minneapolis/St. Paul, investigators confirmed the continued risk due to elevated indoor concentrations of VOCs (Sexton *et al.*, 2004). Pollutant concentrations were compared to risk thresholds established in Minnesota. Median indoor concentrations for benzene and chloroform exceeded the level for a 70-year lifetime cancer risk of 1 in 100,000. The 90<sup>th</sup> percentile concentrations exceeded the cancer risk level for benzene, chloroform, and *p*-dichlorobenzene. The identified pattern of personal VOC concentrations exceeding indoor VOC concentrations, which in turn exceed outdoor VOC concentrations, also was reaffirmed by this study. This relationship held for 13 of the 15 VOCs measured by Sexton *et al.* in Minneapolis/St. Paul over three seasons in 1999.

### Impacts of Attached Garages on Indoor VOC Concentrations

Several studies have investigated the impact that attached garages have on residential indoor levels of VOCs. These studies implicate vehicles as well as other activities (such as storage of chemicals and small engines) within attached garages as the sources of these VOCs. Adgate *et al.* (2004) measured outdoor, indoor, and personal VOC exposures to children in 284 households in Minnesota for ten VOCs. Households with attached garages had significantly higher concentrations of benzene, chloroform, styrene, and *m/p*- and *o*-xylene compared to households without an attached garage. Thomas *et al.* (1993) investigated the temporal variability of benzene exposures for residents in several New Jersey homes with attached



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garages or tobacco smoke. In homes with attached garages or ETS, mean indoor and personal benzene concentrations were two to five times higher than outdoor levels at all but one home. Measured benzene levels in four garages ranged from 3-196  $\mu\text{g}/\text{m}^3$ ; garage source strength estimates ranged from 310-52,000  $\mu\text{g}/\text{hr}$ . The mass transfer rate for benzene from sources in the garage to living areas ranged from 730-26,000  $\mu\text{g}/\text{hr}$ . Material and activities inside the garage were the sources of the benzene.

Several studies have explicitly studied VOC emissions from vehicles in attached garages. Gammage *et al.* (1984) determined that high levels of VOCs indoors in two houses were associated with the operation of a car in a basement garage. Tsai *et al.* (2000) also found elevated indoor concentrations of benzene, toluene, and methanol resulting from automobile emissions released in an attached garage. In a detailed Canadian study, Graham *et al.* (2004) measured the concentration of 175 VOCs (non-methane hydrocarbons and halogenated compounds) inside 16 residences over two seasons while a vehicle in the attached garage was operated under brief cold-start and hot-soak conditions. Positive net changes of indoor VOC concentrations were observed whose signatures were similar to those of the vehicle profile releasing the emissions. Chemical Mass Balance (CMB) modeling suggested that the infiltration of vehicle emissions into the house could account for between 9 and 85% of the measured hydrocarbon concentrations in the house during the test period.

In a study of indoor air samples collected from 137 Anchorage homes between December 1994 and February 1996, the Anchorage Air Pollution Control Agency determined that the presence of an attached garage was the factor most strongly associated with in-home benzene; indoor elevated benzene levels were strongly related to the use of a garage to park vehicles or store fuel or small engines. Other VOCs (toluene, ethylbenzene, *m/p*-xylene, *o*-xylene) were also higher in homes with attached garages; geometric mean concentrations were two to four times higher in homes without attached garages. A regression model for benzene in homes with attached garages revealed a significant impact of a car parked in the garage (vs. no car), number of trips originating from the attached garage, fuel having been opened within the last three days, presence of forced air furnaces (vs. hot water boilers), and living areas above the garage (vs. adjacent to the garage) (*Architectural, Behavioral and Environmental Factors Associated with VOCs in Anchorage Homes*, 1998).

The primary reason that attached garages contribute to elevated indoor levels of CO and VOCs is due to air leakage into the residence through the garage-to-residence interface. Air leakage rates between attached garages and homes have been measured in several different northern climate locales (i.e., Canada, Minnesota) throughout the last decade, with garage-to-residence leakage/infiltration rates ranging from 9% to 17% (*Air Infiltration from Attached Garages in Canadian Houses* 2001, *Evaluation of Homes Built to the New Minnesota Energy Code* 2002, *Garage Performance Testing* 2004). Garage-to-residence infiltration rates may differ in California from these reported values to climatic differences as well as variations in regional building codes.

Several mitigation options may be useful in reducing leakage (Built Green of Colorado, 2004), including aerodynamically decoupling the garage from the house, building ductwork 10 times tighter than the current standard, ventilating garages with either active or passive vents, equipping every home with a CO sensor, testing a subset of houses in a given area to ensure efforts are working, and informing homeowners (Built Green of Colorado, 2004).

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### VOCs in Public and Commercial Buildings

Girman *et al.* (1999) identified VOCs in public and private buildings, but did not associate the presence of VOCs with a particular source. Girman analyzed data collected at 56 office buildings (most with no known indoor air quality problems) across the U.S. Of the 48 VOCs identified in indoor air, eight were found in all of the samples, another 26 were found in 81-99% of the samples. Indoor concentrations of 27 VOCs were at least twice the corresponding outdoor concentrations. The 12 VOCs with the highest median indoor concentrations were acetone, toluene, *d*-limonene, *m*- and *p*-xylenes, 2-butoxyethanol, *n*-undecane; benzene, 1,1,1-trichloroethane, *n*-dodecane, hexanal, nonanal, and *n*-hexane. Only acetone (29  $\mu\text{g}/\text{m}^3$ ) was present at a concentration above 10  $\mu\text{g}/\text{m}^3$ . The study was designed to provide baseline data on VOCs in U.S. office buildings relative to complaint buildings. The author did not compare indoor concentrations to guideline levels; however, levels were below available minimum risk levels.

### 2.3.3 Environmental Tobacco Smoke

Environmental tobacco smoke (ETS) is the complex mixture of thousands of gaseous and particulate compounds emitted from the burning end of cigarettes and cigars, and in smokers' exhaled breath. ETS is also called "second-hand smoke", "passive smoke", and "involuntary smoking". Many chemicals in ETS have been identified as toxic air contaminants, and not surprisingly, ETS is estimated to cause thousands of deaths due to cancer and cardiac disease, and many thousand asthma episodes and other illnesses (ARB/OEHHA, 2004/2005). Cigarettes are the most common source of ETS, but cigars and pipes are also sources.

Passage of a statewide smoke-free workplace law in California in 1995 (AB 13, Labor Code 6404.5) eliminated smoking in nearly all California indoor workplaces—including restaurants, bars and gaming clubs. In 1999, 93.4% of California's indoor workers reported working in a smoke-free environment, compared to only 45% in 1990 (Gilpin *et al.*, 2001). Additionally, the percentage of Californians with children under the age of 18 who do not allow smoking in the household has increased substantially. In 1994, 63% of Californians with children did not allow smoking in the house; by 2001, 78% did not allow it (Gilpin *et al.*, 2001). Because of such laws and trends, California has lower ETS exposures than most other states.

#### 2.3.3.1 Health Effects of ETS

Exposure to ETS has been linked with a variety of adverse health effects, including heart disease, asthma episodes, other respiratory illness, and lung cancer (OEHHA, 1997). Primary tobacco smoke is an established human carcinogen for smokers (IARC, 2002; U.S. DHHS, 1989). ETS has been identified as a cause of lung cancer in nonsmokers (U.S. DHHS, 1986; U.S. EPA, 1992; IARC, 2002). More recently, there is evidence from analyses of epidemiological studies that ETS exposure has a causal association with breast cancer (ARB/OEHHA, 2004/2005).

Table 2.8, below, provides the current estimates of the health impacts of ETS on Californians and Americans every year, taken from the ARB/OEHHA draft report for the identification of ETS as a toxic air contaminant, which is currently in its last stage of scientific review (ARB/OEHHA, 2004/2005). In addition to the large cardiac and lung impacts on adults, ETS has a number of serious impacts on children's health. These include exacerbation of asthma, increased respiratory tract infections, increased middle ear infections, low birth weight, sudden infant death syndrome (SIDs), and developmental impacts (ARB/OEHHA, 2004/2005). Children under 18 months of age have greater tendency to suffer from bronchitis or pneumonia when their parents are smokers. A small reduction in birth weight has been repeatedly observed with

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mothers who are active smokers. Studies have shown fairly consistently that maternal smoking during pregnancy is adversely associated with measures of cognition and behavior in children (NCI, 1999). There is epidemiological evidence of a causal relationship between maternal smoking in general and risk of SIDS.

**Table 2.8 Current Estimated Attributable Risks Associated with ETS**  
(from ARB/OEHHA, 2005)

OUTCOME	EXCESS # IN CA PER YR	EXCESS # IN U.S. PER YR
<b>Pregnancy:</b> <b>Low Birth Weight</b> <b>Pre-term Delivery</b>	1,600 4,700	24,300 <sup>1</sup> 71,900
<b>Cardiac death</b> <b>(Ischemic heart disease death)</b>	1,700-5,500 <sup>2</sup>	22,700 <sup>3</sup> - 69,600
<b>Lung Cancer Death</b>	400 <sup>4</sup>	3400
<b>Asthma episodes</b> <b>(children)</b>	> 31,000 <sup>5</sup>	202,300 <sup>6</sup>
<b>Otitis media visits</b>	51,700 <sup>7</sup>	789,700 <sup>8</sup>
<b>SIDS</b>	21 <sup>9</sup>	431 <sup>10</sup>
<b>Breast cancer</b>	All studies: OR 1.26 (95% CI 1.10-1.45) <sup>11</sup> Best studies: OR 1.90 (95% CI 1.53-2.37) Approximate 26-90% increased risk	

<sup>1</sup> Based on adult females reporting exposure to ETS in NHANES III for 1995 (Pirkle et al., 1996)

<sup>2</sup> Based on California Dept Health Services.

<sup>3</sup> [www.dhs.ca.gov/hisp/chs/OHIR/vssdata/2000data/OOCh5pdf/5\\_9\\_Reorg.PDF](http://www.dhs.ca.gov/hisp/chs/OHIR/vssdata/2000data/OOCh5pdf/5_9_Reorg.PDF). Table 5-9 for yr 2000

<sup>4</sup> Based on Anderson and Arias (2003). National Vital Statistics Report. Vol 51(9) Table 2 for yr 2000  
Ischemic heart diseases including AML.

<sup>5</sup> Assuming California exposure and death rates are similar to national rates and California population is 12% of national population.

<sup>6</sup> Based on number asthma attacks or episodes in previous 12 months for 0-17 year olds. Calculated from California Health Interview Survey for 2001

<sup>7</sup> Based on number asthma attacks or episodes in previous 12 months for 0-14 year olds. CDC-MMWR 2002 51(SS01)

<sup>8</sup> Calculated by applying national value (H6) and assuming 12% of US population lives in California

<sup>9</sup> Based on National Center for Health Statistics Series 13 No. 137. Ambulatory Health Care Visits by Children: Principal Diagnosis and Place of Visit for yrs 1993-1995.

<sup>10</sup> Based on California Dept Health Services.

<sup>11</sup> [www.dhs.ca.gov/hisp/chs/ohir/vssdata/2000data/OOch4pdf/8reorg.pdf](http://www.dhs.ca.gov/hisp/chs/ohir/vssdata/2000data/OOch4pdf/8reorg.pdf). Table 4-8 for yr 2000

<sup>12</sup> Based on National Center for Health Statistics. [www.cdc.gov/nchs/fastats/infort.htm](http://www.cdc.gov/nchs/fastats/infort.htm) for yr 2000

LBW = low birth weight; N/A = data not available.

<sup>13</sup> OEHA is unable at this time to calculate an attributable risk as it is not possible to account accurately for the portion attributable to other known risk factors.

ETS affects many tissues and organs of the body. ETS is causally associated with coronary heart disease in smokers and nonsmokers (NCI, 1999). Research suggests that chronic ETS exposure may increase the risk of stroke by about 82% (Bonita *et al.*, 1999). ETS is estimated to cause about 400 excess deaths due to lung cancer each year in California (ARB/OEHHA, 2004/2005). In reviewing the literature on asthma and indoor air, the Institute of Medicine (2000) found that:

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“There is sufficient evidence to conclude that there is an association between ETS exposure and the development of asthma in younger children. [However], there is inadequate or insufficient evidence to determine whether an association exists between ETS exposure and the development of asthma in school-aged or older children, or in adults. There is sufficient evidence to conclude that there is a causal relationship between ETS exposure and exacerbations of asthma in preschool-aged children. There is limited or suggestive evidence of an association between chronic ETS exposure and exacerbations of asthma in older children and adults. Limited or suggestive evidence of an association between acute ETS exposure and exacerbations also exists for asthmatics sensitive to this exposure”.

Eye and nasal irritation are the most commonly reported symptoms among adult nonsmokers exposed to ETS (ARB/OEHHA, 2004/2005).

Nazaroff and Singer (2004) estimated mean daily exposure to and inhalation intake of specific hazardous air pollutants found in ETS for non-smokers who live with smokers. The approach used a model based on cigarette consumption patterns, emission factors, residence characteristics, and population statistics. Results indicate “the ratio of estimated average exposure concentrations to reference concentrations is close to or greater than one for acrolein, acetaldehyde, 1,3-butadiene, and formaldehyde, indicating potential for concern regarding noncancer health effects from chronic exposure. In addition, lifetime cancer risks from residential ETS exposure are estimated to be substantial (~2 to 500 per million) for each of five known or probable human carcinogens: acetaldehyde, formaldehyde, benzene, acrylonitrile, and 1,3-butadiene.”

### 2.3.3.2 ETS Sources

The variability in emissions between brands of cigarettes is relatively low, though emissions from cigars and cigarettes vary in magnitude. Daisey *et al.* (1998) conducted a chamber study testing six of the most popular commercial brands in California and one reference cigarette for emissions of 21 different air toxics and other airborne compounds, including VOCs, nicotine, aldehydes, and airborne particulate matter (estimated to be PM<sub>2.5</sub>). Diluted sidestream smoke (produced by a smoking machine that smoked three cigarettes sequentially) was used to approximate ETS aging in a room-sized chamber, and a mass-balance model was used to generate estimates of indoor concentrations. Among the VOCs, acetaldehyde and formaldehyde displayed the highest emission factors (average emission factors 3,340 ng/mg tobacco and 2,040 ng/mg tobacco, respectively), and PM showed an emission factor of 12,400 ng/mg. The nicotine emission factor was 1,410 ng/mg tobacco. These results suggest that ETS has a substantial influence on indoor concentrations of these compounds.

Chuang *et al.*, (1999) investigated children’s exposure to polycyclic aromatic hydrocarbons (PAHs) in low-income rural and inner-city areas in North Carolina. Indoor/outdoor air samplers and real-time PAH monitors were used to obtain measurements. The researchers determined that potentially carcinogenic PAH concentrations were significantly higher in smokers’ homes than in non-smoking homes (geometric mean: 6.14 ng/m<sup>3</sup> vs. 1.38 ng/m<sup>3</sup>, respectively). Additionally, the authors discovered that children in both rural and inner-city homes received higher potential doses of PAHs than adults, in part due to their lower body weights.

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### 2.3.3.3 ETS Concentrations

Restrictions on smoking in California from the late 1980s to mid 1990s in workplaces and in public locations such as restaurants, bars, and gaming clubs have led to a reduction in smoking in indoor environments in California, with commensurate reductions in indoor concentrations of ETS and non-smokers' exposure levels. A number of studies published since 1996 have shown that ETS constituents are present at lower concentrations in public places following smoking bans than they were prior to the bans (Ott *et al.*, 1996; Hammond, 1999; Switzer *et al.*, 2001; Repace, 2003), and that levels can be considerably higher in smoking versus comparable nonsmoking areas (Glasgow *et al.*, 1998; Hammond, 1999; Graves *et al.*, 2000).

Despite California's workplace smoking ban, high indoor ETS concentrations still can be found in smokers' homes and in private vehicles (Ott *et al.*, 2003; Park *et al.*, 1998; Offermann *et al.*, 2002). Children's exposure to ETS is greatly impacted in these two environments when in the presence of a smoking parent or other adult. Children spend up to 85% of their time in the home (Wiley *et al.*, 1991a). Thus, the potential for exposure to ETS can be extremely high when smoking occurs in a child's home. As of 2001, 22% of smoking parents still allowed smoking inside the home (Gilpin *et al.*, 2001). Likewise, exposure in vehicles can be quite high due to the presence of a strong source in a relatively small volume of air. Recent residential respirable particulate matter (RSP, PM3.5) measurement is limited to a single study (Ott *et al.*, 2003). A level of 300  $\mu\text{g}/\text{m}^3$  was measured in the bedroom where one cigarette was smoked; 5,500  $\mu\text{g}/\text{m}^3$  was the maximum bedroom level when three cigarettes were smoked. RSP levels ranging from 92  $\mu\text{g}/\text{m}^3$  (with ventilation) to 1,195  $\mu\text{g}/\text{m}^3$  (without ventilation) have been measured inside a minivan (Offermann *et al.*, 2002).

Three comprehensive review documents summarize nicotine and RSP concentrations measured in smoking environments prior to 1996 (U.S. EPA, 1992; Guerin *et al.*, 1992; NCI, 1999). Comparison of mean nicotine concentrations from these earlier reviews with data published after 1995 reveals that the means have decreased in workplaces and restaurants, but less so in homes. In studies conducted before 1996, mean nicotine concentrations in offices and restaurants ranged from about 1 to 36  $\mu\text{g}/\text{m}^3$ . In a more recent review, Hammond (1999) reported means of 2 to 8  $\mu\text{g}/\text{m}^3$  for these locations. Also, according to the Hammond review, nicotine levels were two- to six-times lower in indoor workplaces with smoking bans than in offices that allowed smoking (less than 1  $\mu\text{g}/\text{m}^3$  vs. 2-8  $\mu\text{g}/\text{m}^3$ , respectively). It appears that nationally, as smoking has become a less accepted social behavior, individuals are not smoking in indoor public locations as much as they did a few years earlier (in California, smoking is prohibited in indoor workplaces).

Levels of RSP are generally comparable in both older and newer studies, but slightly lower in the newer studies (relative to 1996). Studies highlighted in the NCI review (1999) reported RSP concentrations consistent with other reviews. All measured levels tend to range from about 100 to 400  $\mu\text{g}/\text{m}^3$  in offices and restaurants that allow smoking. Switzer *et al.*, (2001) measured RSP levels at a church bingo site of 87 to 348  $\mu\text{g}/\text{m}^3$  above outdoor levels, and at less than 15  $\mu\text{g}/\text{m}^3$  when smoking was banned. Similarly, PM3.5 concentrations at a sports tavern in California were 56.8  $\mu\text{g}/\text{m}^3$  with smoking, and 5.9 – 12.9  $\mu\text{g}/\text{m}^3$  with smoking banned (Ott *et al.*, 1996). Similar to nicotine levels, these recent RSP data from smoking locations are somewhat lower than the pre-1996 data. RSP levels are much lower (<15  $\mu\text{g}/\text{m}^3$ ) in indoor locations where smoking is prohibited.

The majority of California homes never or rarely contain smokers, and would typically have a nicotine level lower than 0.5  $\mu\text{g}/\text{m}^3$ . However, the recent body of data indicates that those who

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choose to smoke in their home have remained consistent in their smoking patterns over the years. It is important to note that concentrations of lower volatility ETS components such as nicotine, 3-ethenylpyridine, phenol, cresols, and naphthalene accumulate on surfaces to the extent that their re-emission (when a smoker is not present) is an important route of indirect exposure for non-smokers (Singer et al., 2003; Singer et al., 2002).

Table 2.9 summarizes current estimates of indoor levels of nicotine and RSP for California. A more detailed discussion of indoor ETS concentrations is provided in the Draft *Technical Support Document – Part A, Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant* (ARB/OEHHA, 2004/2005).

**Table 2.9. Estimates of Current California Indoor Concentrations of Nicotine and Respirable Suspended Particulates (RSP)**

Environment	Nicotine Concentration $\mu\text{g}/\text{m}^3$	RSP Concentration $\mu\text{g}/\text{m}^3$
Homes with smokers present	0.5 – low <sup>1</sup> 3.0 – medium 6.0 – high	300-5,500 <sup>3</sup>
Offices/public buildings With smoking Smoking prohibited	2 – 8 <sup>2</sup> <1	56.8 – 348 <sup>4</sup> <15
Vehicles With ventilation Without ventilation	NA NA	~100 <sup>5</sup> ~1,200

1. Glasgow *et al.*, 1998
2. Hammond, 1999
3. Ott *et al.*, 2003
4. Ott *et al.*, 1996 and Switzer *et al.*, 2001
5. Offermann *et al.*, 2002

### 2.3.4 Biological Contaminants

Biological contaminants are substances of plant, animal, or microbial origin. They are naturally abundant in the outdoor and indoor environments, but are considered contaminants when found in undesired locations or at elevated concentrations. They include bacteria, viruses, and fungi; allergens from animal dander, pollen, fungi, and the fecal particles or body fragments from house dust mites and cockroaches; as well as chemicals emitted by mold and bacteria such as endotoxins and mycotoxins. Exposure to biological contaminants may cause a variety of health effects, including asthma, allergy, infection, irritation, and toxic responses. Building related illness, or BRI, is an illness for which the specific cause—usually a virus, bacteria, or fungi—can be identified within the building. Examples include Legionnaire's disease, caused by the *Legionella* bacterium, or humidifier fever.

#### 2.3.4.1 Health Effects of Biological Contaminants

The health effects of biological contaminants can be grouped into three major categories:

- Communicable disease transmission: Many infectious diseases are transmitted from person to person in indoor air. For example, inhalation of viruses is associated with influenza, measles, and chicken pox. The primary mode of transmission for the common cold virus has

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not been proven conclusively. Studies have shown transmission by both inhalation and direct or indirect contact between infected droplets from coughing or sneezing onto mucous membranes of the eyes, nose or mouth (Goldmann, 2002). Inhalation of bacteria is associated with tuberculosis and Legionnaire's disease. Epidemiological studies have often found significantly lower prevalence of respiratory illness or surrogates for respiratory illness (sick leave, total absence from school) in buildings with higher ventilation rates, reduced office sharing, and less crowding (Fisk, 2000; Myatt *et al.*, 2004; Shendell *et al.*, 2004). These and other studies indicate that a significant portion of common respiratory illness may be transmitted indoors by airborne particles; however, the proportion of total disease transmission that occurs through this route cannot be easily quantified.

- Hypersensitivity reactions: Many biological agents can provoke a hypersensitivity response in individuals who are genetically predisposed to developing allergic disorders. Allergic rhinitis (hay fever) and allergic asthma are the most common examples of hypersensitive responses to biological contaminants. Common symptoms and signs are watery eyes, runny nose, sneezing, nasal congestion, itching, coughing, wheezing, difficulty breathing, headache, and fatigue. Allergens from fungal spores, house dust mites, cockroaches, dog and cat dander, and pollen are frequently found indoors. When high concentrations of these allergens are present indoors they can trigger allergic responses or asthmatic exacerbation. Hypersensitivity pneumonitis is a rare immune-mediated lung disorder initially found in farmers and workers exposed to high doses of organic dusts in agricultural or industrial settings. A few cases of this disease have been attributed to indoor exposure to bacteria or mold growth. Only susceptible persons exposed to large amounts of these allergens are at risk of developing hypersensitivity pneumonitis (IOM, 2004).
- Toxic responses: Many individuals in persistently damp or moldy buildings report symptoms such as headache, memory difficulties, vomiting, diarrhea, and increased frequency of cold/flu illnesses that do not appear to be caused by allergic or infectious mechanisms. The causes of such symptoms have not been identified. Some researchers have postulated that exposure to biological toxins such as endotoxins (components of the outer membrane of Gram-negative bacteria) and mycotoxins (secondary metabolic products of some fungi) may induce such symptoms. However, the health impact of inhalation exposure to biological toxins in indoor environments is not well understood.
- More detailed information on the health effects of mold is discussed below in Section 2.3.4.3.

### 2.3.4.2 Sources of Biological Contaminants

Biological contaminants include allergens such as animal dander, house dust mites, cockroaches, and pollen; bacteria, viruses, and fungi; and chemicals emitted by mold and bacteria such as endotoxins and mycotoxins.

#### Animal Dander

Dogs and cats are kept as pets in 32 and 27% of U.S. households, respectively (AVMA, 1997). Allergy to cats is reported to be about twice as common as allergy to dogs. Dog and cat allergens are found on small particles that can remain airborne for prolonged periods. The particles also adhere readily to fabrics such as clothing, upholstered furniture, and carpet and can easily be carried from animal-owning homes into offices, schools and day care centers (Custovic *et al.*, 1998). In a recent nationally representative study, both dog and cat allergens were detected in all U.S. homes (Arbes, 2004). Asthmatics who are sensitive to cat allergen

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may experience allergic symptoms (congestion, runny nose, itching), asthma exacerbation and compromised lung function (chest tightness or wheezing) at cat allergen levels typically found in schools and homes without cats (Bollinger *et al.*, 1996).

### House Dust Mites

House dust mites are microscopic relatives of spiders that feed on human skin cells and other organic material. Primary mite allergens are concentrated in their fecal particles and body fragments. These allergens are very potent, but concentrations in indoor air typically are low because the allergens are attached to larger particles (at least 10  $\mu\text{m}$ ) that settle rapidly. Consequently dust mite allergens are found predominantly in carpets, pillows, bedding, and upholstered furniture. In locations where humidity is high for most of the year, dust mites produce larger quantities of allergen (Rosenstreich *et al.*, 1997). In contrast, mites cannot survive in desert or mountain (5,000 feet elevation or higher) regions where indoor humidity is routinely low. In a recent study of U.S. homes, mite allergens were found in all beds tested. 46 percent of these homes had mite allergen at levels previously associated with allergic sensitization, while 24% had levels associated with an increased risk for asthma attacks in asthmatics allergic to dust mites (Arbes *et al.*, 2003).

### Cockroaches

While many insects have been implicated as sources of inhaled allergens in small indoor studies, cockroaches are the only insects that have routinely been identified as a common source of indoor allergens (IOM, 2000). The exact part of the cockroach responsible for its allergens is unknown but may involve fecal particles and body parts. Like house dust mites, cockroach allergen is associated with particles larger than 5  $\mu\text{m}$  and these particles become airborne only when settled dust is disturbed. Cockroach activity frequently occurs in indoor areas where food or standing water is available, such as kitchens and bathrooms. Cockroach hypersensitivity is highest among the urban poor, but the complex interrelationship between race, poverty and residence has been difficult to resolve (IOM, 2000). Exposure to cockroach allergen has been associated with almost a two-fold increased risk of wheeze in infants less than one year old (Belanger *et al.*, 2003). In a recent study of southern California children, asthma diagnosis before five years of age was associated with exposures in the first year of life to cockroaches and other environmental agents (Salam *et al.*, 2003).

### Endotoxins

Endotoxins are components of the outer membranes of Gram-negative bacterial cells. These bacteria occur naturally on plants and are abundant in soil and in the human intestinal tract. Endotoxins are released when these bacteria die or their cell membranes are damaged; thus, these toxins are always found in the outdoor environment. High-level exposures to airborne endotoxins in agricultural and waste-disposal industries contribute to acute and chronic bronchitis that may lead to decreased lung function (Vogelzang *et al.*, 1998). In indoor environments, lower airborne endotoxin concentrations have been associated with both adverse health effects (increased asthma symptoms and medication use) and protective health effects (decreased prevalence of allergy) (IOM, 2004). Many factors appear to interact to modulate health effects associated with endotoxin exposure (Song *et al.*, 2003).

### Viruses

Viruses are the smallest and simplest infectious agents, unable to survive well outside their plant, animal or human host. In outdoor air, viral survival depends on season, moisture content and temperature of the air, wind conditions, sunlight and presence of atmospheric pollutants (Cox, 1995). In the indoor environment, the infectivity of airborne viruses is affected by factors such as relative humidity and room temperature (Otten *et al.*, 1999). Most viral infections are



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spread from person to person in the indoor environment. Rarely a virus such as Hantavirus (also known as Sin Nombre or Four Corners virus), endemic in some populations of deer mice in the Southwestern United States, can be transmitted to humans. Indoor air exposure to this virus can occur when housecleaning activities disturb dust or nests resulting in aerosolized mouse saliva or excreta (Mills, 2002).

### Damp buildings

Moisture is common in buildings, with most studies reporting signs of dampness in at least 20% of homes examined (IOM, 2004). Moisture problems originate from rainwater, groundwater, plumbing, construction, water use by occupants and condensation of water vapor. Although some moisture is present in all buildings, excessive dampness is more likely to occur in buildings that are older, lack central heating, are poorly insulated and overcrowded (IOM, 2004). The assessment of building dampness is complicated by the absence of a generally accepted definition of “dampness” or what constitutes a “dampness problem”.

A recent report by the National Academy Institute of Medicine, entitled *Damp Indoor Spaces and Health* (IOM, 2004), reviewed the scientific literature regarding indoor dampness and its relationship to the various health outcomes that have been attributed to damp or moldy indoor environments. Key findings from this report are shown in Table 2.10. The report concludes that although many details require clarification through future research, the currently available scientific evidence is sufficient to regard excessive indoor dampness as a health threat to building occupants.

A review of the medical literature has shown that occupants of damp buildings are twice as likely to experience coughing, wheezing and asthma attacks as those in dry buildings (Bornehag *et al.*, 2001). The underlying causes and mechanisms of these illnesses are not completely understood. Some studies have associated occupant health problems with fungal or bacterial growth on building materials such as drywall and carpeting (Husman, 1996; Verhoeff and Burge, 1997; Peat *et al.*, 1998). However, building dampness is also known to increase emission of volatile organic compounds (VOCs) from decomposition of flooring materials, even without microbial growth (Pasanen *et al.*, 1998). Damp concrete floors are known to increase the rate of chemical degradation of polyvinyl chloride floor coatings and glues, resulting in emissions of ammonia and other VOCs into the indoor air (Gustavsson and Lundgren, 1997; Wiglusz *et al.*, 1998; Tuomainen *et al.*, 2004). Damp buildings also encourage the growth and allergen production capacity of cockroaches, house dust mites and other arthropods, as well as the survival of respiratory viruses.

#### 2.3.4.3 *Indoor Mold*

The remainder of this section discusses indoor mold in detail, because of its increasing occurrence at problem levels in indoor environments.

Molds are simple, microscopic organisms, present virtually everywhere, indoors and outdoors. Molds, along with mushrooms and yeasts, are fungi, which play a critical role in nature by breaking down dead plant and animal matter and recycling nutrients in the environment. For molds to grow and reproduce, they need only moisture and a food source – organic plant material, such as leaves, wood or paper or animal products such as leather. Because molds grow by digesting organic material, they gradually destroy whatever they grow on. Visible mold growth on cloth or building materials or furnishings, sometimes referred to as “mildew”, often has a wooly or cottony appearance that is frequently green, gray, brown, or black but may also

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be white or a range of other colors. Many molds that can grow indoors release countless tiny, lightweight spores that travel easily through the air.

### Health Effects of Mold

In recent years, media attention to indoor mold has surged, leading to rising concern about mold-related health effects. Because mold spores are ubiquitous in air or dust, everyone contacts them on a daily basis, usually without evident harm. However, persons who are allergic to mold and those with compromised immune systems may develop serious health problems from exposure to routine types and amounts of indoor mold.

**Table 2.10. Health Outcomes and Indoor Dampness<sup>1</sup>**

<b>Sufficient Evidence of a Causal Relationship</b> <ul style="list-style-type: none"><li>• None (the evidence was not sufficient to link dampness as a clear cause of any health outcome)</li></ul>
<b>Sufficient Evidence of an Association</b> <ul style="list-style-type: none"><li>• Upper respiratory tract (nasal and throat) symptoms</li><li>• Asthma symptoms in sensitized asthmatic persons</li><li>• Wheeze</li><li>• Cough</li></ul>
<b>Limited or Suggestive Evidence of an Association</b> <ul style="list-style-type: none"><li>• Shortness of breath</li><li>• Lower respiratory illness in otherwise healthy children</li><li>• Asthma development</li></ul>
<b>Inadequate or Insufficient Evidence to Determine Whether an Association Exists</b> <ul style="list-style-type: none"><li>• Airflow obstruction in otherwise-healthy persons</li><li>• Skin symptoms</li><li>• Chronic obstructive pulmonary disease</li><li>• Mucous membrane irritation syndrome</li><li>• Inhalation fevers (non-occupational exposures)</li><li>• Lower respiratory illness in otherwise healthy adults</li><li>• Rheumatologic and other immune diseases</li><li>• Acute idiopathic pulmonary hemorrhage in infants</li><li>• Gastrointestinal tract problems</li><li>• Fatigue</li><li>• Neuropsychiatric symptoms</li><li>• Cancer</li><li>• Reproductive effects</li></ul>

1. Not applicable to immunocompromised persons, who are at increased risk for fungal infections.  
Source: IOM, 2004

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Fungi produce a very large number of allergens, with each fungus potentially producing several different allergenic compounds depending on its growth pattern and environmental conditions. More than 80 fungal species have been associated with allergic diseases (Day *et al.*, 2001). Unfortunately, the limited number of standardized materials available to allergists to test patients for mold allergies complicates our ability to determine the frequency of mold allergy in U.S. or California populations. Humans are known to potentially encounter approximately 200 different species of fungi outdoors and perhaps 50 species indoors. Typical symptoms that mold-allergic persons report (alone or in combination) include: respiratory problems, such as wheezing, difficulty breathing and shortness of breath; nasal and sinus congestion; eye irritation (burning, watery or reddened eyes); dry, hacking cough; nose or throat irritation; and skin rashes. In rare instances exposures to fungi may illicit more intense immunological responses such as allergic bronchopulmonary aspergillosis and allergic fungal sinusitis. These conditions involve fungal colonization of the airways or sinuses (Storey *et al.*, 2004).

Serious lung infections from a few fungal groups such as *Aspergillus* and *Fusarium* species are common in persons being treated with high-dose cancer chemotherapy, recent solid-organ transplant recipients or those who are otherwise immunocompromised (Summerbell, 2001). Children in damp or moldy buildings sometimes report having more respiratory infections, including colds and ear infections (Rylander, 2000). Some investigators have suggested that this increase is due to an immunosuppressive effect of exposure to indoor fungal growth (Johanning *et al.*, 1996). While some fungi have been shown to cause immune suppression in experiments with laboratory animals, it is not yet clear if damp or moldy building exposures can cause significant changes in the human immune system.

Organic dust toxic syndrome is a general term used in reference to illnesses related to inhalation of bacterial endotoxins or fungal toxins, typically resulting from occupational exposures to bioaerosols. The symptoms are similar to those of hypersensitivity pneumonitis (Storey *et al.*, 2004). One other possible health outcome from exposure to mycotoxins is pulmonary hemorrhage (Storey *et al.*, 2004).

In studies of health effects associated with indoor fungal exposure, some people without allergies report respiratory and other symptoms similar to those experienced by mold-allergic individuals. In addition, occupants of moldy buildings have reported some health outcomes that are not usually associated with allergy (such as memory loss, depression, fatigue, mood swings, and “hemorrhage in the mucous membranes of the intestinal and respiratory tracts”) (IOM, 2004). Investigators are exploring whether these effects are associated with exposure to one or more fungal constituents such as:

- Fungal toxins – chemicals known to be produced under certain fungal growth conditions by more than 400 fungal species and capable of producing a toxic response in animals or other microbes.
- Some structural components of fungi – for example, glucans (chemicals that make up all fungal cell walls) may affect the activity of immune cells in the lung.
- Microbial volatile organic compounds – gaseous substances that account for the odors identified when mold is growing indoors. While often associated with respiratory symptoms in damp buildings, the specific contribution of these compounds to health complaints is still unclear.

Key findings regarding indoor mold growth and health outcomes from the National Academy Institute of Medicine report, *Damp Indoor Spaces and Health* (IOM, 2004), are summarized in Table 2.11. The report concludes that although more research is needed to define the role of molds, mycotoxins and other fungal components, as well as the potential for synergistic

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interaction between molds and other microbial or chemical agents in damp buildings, the currently available scientific evidence is sufficient to conclude that indoor mold is associated with upper respiratory symptoms, cough, wheeze, asthma symptoms in sensitized asthmatic individuals and, rarely, hypersensitivity pneumonitis in susceptible persons.

**Table 2.11. Presence of Indoor Mold and Health Outcomes<sup>1</sup>**

<b>Sufficient Evidence of Causal Relationship</b> <ul style="list-style-type: none"><li>• None (the evidence was not sufficient to link mold as a clear cause of any health outcome)</li></ul>
<b>Sufficient Evidence of an Association</b> <ul style="list-style-type: none"><li>• Upper respiratory tract (nasal and throat) symptoms</li><li>• Asthma symptoms in sensitized asthmatic persons</li><li>• Wheeze</li><li>• Cough</li><li>• Hypersensitivity pneumonitis (a rare immune-mediated lung condition) in susceptible persons<sup>2</sup></li></ul>
<b>Limited or Suggestive Evidence of an Association</b> <ul style="list-style-type: none"><li>• Lower respiratory illness in otherwise healthy children</li></ul>
<b>Inadequate or Insufficient Evidence to Determine Whether an Association Exists</b> <ul style="list-style-type: none"><li>• Shortness of breath</li><li>• Airflow obstruction in otherwise healthy persons</li><li>• Skin symptoms</li><li>• Asthma development</li><li>• Chronic obstructive pulmonary disease</li><li>• Mucous membrane irritation syndrome</li><li>• Inhalation fevers (non-occupational exposures)</li><li>• Lower respiratory illness in otherwise healthy adults</li><li>• Rheumatologic and other immune diseases</li><li>• Acute idiopathic pulmonary hemorrhage in infants</li><li>• Gastrointestinal tract problems</li><li>• Fatigue</li><li>• Neuropsychiatric symptoms</li><li>• Cancer</li><li>• Reproductive effects</li></ul>

1. Not applicable to immunocompromised persons, who are at increased risk for fungal infections.

2. For mold or bacteria in damp indoor environments.

Source: IOM, 2004

### Mold Concentrations

It is common in indoor air quality investigations to find mold spores in the air and dust inside homes, with most of these originating outdoors. Many studies have tried to differentiate

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buildings with problem mold growth from non-moldy buildings by evaluating the type and concentration of indoor airborne fungal spores. The concentrations and types of mold spores in indoor air typically are directly related to those in outdoor air. If there is a serious mold problem in a building, the indoor types and concentrations of mold may, or may not, differ from those outdoors at the time of sampling. Numerous studies have found that spore concentrations vary widely in both outdoor and indoor air. Outdoor concentrations vary with geographical location, climate, season, relative humidity, wind direction and types of vegetation in the immediate area of the sampling device (IOM, 2004). Outdoor airborne mold spore concentrations may reach levels as high as 10,000 spores/m<sup>3</sup> (Mullins, 2001). Indoor mold spore concentrations are usually lower than corresponding outdoor levels, but are also quite variable, ranging from 0-10,000 colony forming units/m<sup>3</sup> (Shelton *et al.*, 2002).

Different types of molds are identified and their concentrations measured either by directly examining a sample under a microscope or by culturing the spores and allowing them to grow into colonies that are then counted. There are many reliable methods for collecting and analyzing fungi although no single method can identify all the fungi present in environmental samples (AIHA, 1996; ACGIH, 1999). Thus, different sample collection and analysis techniques often lead to different fungal count or concentration estimates. No standard method of mold identification or spore counting has been proven effective in a wide range of building applications or is mandated for environmental assessment by any federal or California state government agency.

In a recent review of studies aimed at identifying buildings with mold problems, an expert panel of the Institute of Medicine concluded "...fungal counts alone provide little information about the microbial status of an indoor environment" (IOM, 2004). Currently, government and professional industry groups recommend that building investigations for mold include a thorough visual inspection of the premises, documentation of visibly moldy areas, and the use of professional judgment in determining whether mold sampling is appropriate for that particular investigation (U.S. EPA, 2001b; AIHA, 1996; ACGIH, 1999; Miller, 2001).

### 2.3.5 Pesticides

Pesticides can be naturally occurring or synthetic chemicals designed to control and kill insects, weeds, and disease carrying organisms in the home and surrounding landscaping. Ninety% of American homes use pesticides (Gurunathan *et al.*, 1998). Schools commonly apply pesticides in or around the classroom (Addiss *et al.*, 1999; Kaplan *et al.*, 1998; Volberg *et al.*, 1993). Pesticides can be tracked in on clothing or drift in from outdoors, later becoming resuspended in air and accumulating in dust.

#### 2.3.5.1 Health Effects of Pesticides

Pesticides differ in their levels of toxicity and mechanisms of action. Some classes of insecticides are highly toxic, especially to the nervous system. Pesticides with relatively low acute toxicity are not necessarily safe, as they may have the potential to cause cancer or other chronic effects. Other health effects of pesticide exposure include irritation of the skin and eyes, and hormone or endocrine disruption.

Two classes of widely used insecticides in the U.S. are the organophosphates and pyrethroids; both are neurotoxins. Pesticide workers have experienced nausea, headaches, dizziness, and general weakness after exposure to agricultural organophosphates. Typical indoor air concentrations of organophosphates previously approved for home use (i.e., chlorpyrifos and

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diazinon) generally do not result in these symptoms. As a class, pyrethroids are less toxic than organophosphates. Their neurotoxic effects have not been reported for humans exposed to typical levels in the home. Pesticides are often measurable in house dust and carpet dust; levels of contamination are discussed later in this section. The effects of pesticides on children are a particular concern because their behavior can lead to greater exposure than to an adult. Children spend time on the floor where they contact dust that may contain pesticides. The hand-to-mouth behavior of young children may lead to ingesting pesticides. Furthermore, research has shown that low-level chronic exposure to organophosphates can adversely affect children's developing nervous systems (Eskenazi *et al.*, 1999); chronic exposure to pyrethroids has been linked to possible hormonal disruption (Landrigan *et al.*, 1999). There is insufficient evidence to determine if pesticides cause or exacerbate asthma (IOM, 2000).

### 2.3.5.2 Sources of Pesticides

Chlorpyrifos and diazinon, two organophosphate insecticides, were the most widely used pesticide ingredients in common household ant and roach killers and lawn-care sprays (U.S. EPA, 2001a; U.S. EPA, 2000d). The U.S. EPA banned the indoor use of chlorpyrifos and diazinon for non-agricultural settings in December 2000 and March 2001, respectively (U.S. EPA, 2001a; U.S. EPA, 2000d). Chlorpyrifos is persistent in the environment and has been observed in many indoor house dust samples since it was banned.

Other pesticides that are persistent in the environment and have been banned for some time include, DDT, and Dieldrin. DDE is a breakdown product of DDT, a widely used insecticide that was banned in 1972 (U.S. EPA, 2000f). Environmental sources of DDE include soil, atmospheric dispersion, sediment runoff, contaminated plants and animals, and improper use and disposal. Dieldrin was widely used from 1950 to 1974 to control insects on cotton, corn and citrus crops (U.S. EPA, 2000g). Also, dieldrin was used to preserve wood, control termites, and control locusts and mosquitoes. Most uses of dieldrin were banned in 1987. Environmental sources of dieldrin include soil surrounding wooden structures treated for termites; soil or sediment; improper use or disposal; contaminated fish and shellfish; and contaminated dairy products and meat.

Since the ban on indoor use of chlorpyrifos, a class of insecticides called pyrethroids has been used as substitutes for chlorpyrifos and other organophosphate pesticides. Permethrin, the most common pyrethroid, acts on a broad spectrum of insects, and is less persistent than chlorpyrifos in dust and soils.

### 2.3.5.3 Pesticide Concentrations

To date, only one published study has examined indoor pesticide concentrations in California homes. In September 1992, Bradman *et al.* (1997) measured pesticide levels in house dust samples from five farmworker homes and five non-farmworker homes in an agricultural area south of Fresno. This study was conducted before the ban on indoor use of chlorpyrifos and diazinon. However, it cannot be determined if the indoor levels were from infiltration of outdoor air and soil track-in, or from the use of indoor pesticide. Of the ten pesticides detected in house dust, only chlorpyrifos and diazinon levels frequently exceeded 1 µg/g of dust in the homes. In general, farmworker homes had higher levels of chlorpyrifos and diazinon than non-farmworker homes. The highest detectable level of pesticide in non-farmworker homes was 2.5 µg/g of diazinon, and the highest in farmworker homes was 169 µg/g of diazinon.

During 2001-2002 (after the ban on indoor use of chlorpyrifos and diazinon), ARB and DHS funded a comprehensive statewide study of the environmental health conditions in portable

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(relocatable) classrooms. As part of the study, investigators summarized floor-dust pesticide concentration data for twenty different pesticides. Chlorpyrifos, *cis*- and *trans*-permethrin, *ortho*-phenylphenol, piperonyl butoxide (PBO), and esfenvalerate were detected in over 80% of the classrooms. Excluding *ortho*-phenylphenol, these pesticides measured 95<sup>th</sup> percentiles above 1 µg/g. Portable classrooms did not differ significantly from traditional classrooms in their mean pesticide concentrations (Whitmore *et al.*, 2003).

The pesticides detected in California and their concentrations are similar to those found in other studies in other areas of the U.S. A summary of indoor and personal air concentrations and house dust levels is presented in Table 2.12. The following paragraphs provide a brief discussion of some of the table content.

The Non-Occupational Pesticide Exposure Study (NOPES) examined inhalation exposures for 32 different pesticides in 208 residences in Jacksonville, Florida, and 101 residences in Springfield/Chicopee, Massachusetts, over three different seasons from 1986 to 1988 (Whitmore *et al.*, 1994). Seven pesticides – chlordane, chlorpyrifos, diazinon, dichlorvos, heptachlor, *ortho*-phenylphenol, and propoxur – had the highest mean concentrations across seasons in Jacksonville for both indoor and personal air. Mean concentrations of all detectable pesticides captured in carpet ranged from 0.01 µg/g to 15.4 µg/g. The mean concentrations for chlorpyrifos and diazinon found captured in the carpet were 5.8 µg/g and 1.7 µg/g, respectively.

During the 1992 spray season, Simcox *et al.* (1995) vacuum-sampled house dust for four organophosphorus (OP) insecticides, including chlorpyrifos, in 59 homes in Wenatchee, Washington (48 agricultural, 11 non-farming families). All four compounds were detected in 62% of households. Pesticide levels were significantly lower in non-farming homes than in agricultural homes (Table 2.12). OP levels ranged from non-detectable to 17 µg/g in agricultural homes and non-detectable to 0.82 µg/g in non-farming homes. Two-thirds of the agricultural homes contained pesticide levels above 1 µg/g for at least one OP compound.

During May to September 1997, the Minnesota Children's Pesticide Exposure Study (MNCPEs) characterized indoor and personal air levels for children 3 to 12 years old in 102 homes in Minneapolis/St. Paul, Minnesota (Clayton *et al.*, 2003). Although the median values for indoor air were slightly higher than personal air (Table 2.11), there was a highly significant correlation between personal air and indoor air samples for the organophosphates chlorpyrifos (0.81), malathion (0.51), and diazinon (0.62). Air levels for chlorpyrifos and diazinon were measurable for >90% and >65% of the participants, respectively. Permethrin was also detectable in air with a median concentration of <0.2 ng/m<sup>3</sup>.

In the National Human Exposure Assessment Survey (NHEXAS) performed in Arizona, Gordon *et al.* (1999) measured residential levels of chlorpyrifos and diazinon. Chlorpyrifos was found in dust more often than diazinon (88% vs. 53%), and had nearly the same occurrence in indoor air as diazinon (65% vs. 63%). There was a small association between chlorpyrifos in dust and air (Pearson = 0.096, Spearman = 0.773). Although the NHEXAS results are generally comparable to those from other studies, Gordon *et al.* (1999) found a higher maximum level for pesticides in residential samples than previously reported.

In a recent study, Rudel *et al.* (2003) analyzed 39 air and 38 dust samples (collected by vacuum cleaner) for pesticides in 120 homes in Cape Cod, Massachusetts. Of the 23 pesticides detected in air and 27 detected in dust, chlorpyrifos was one of the most abundant with 90<sup>th</sup> percentile concentrations of 12 ng/m<sup>3</sup> in air and 1.87 µg/g in dust. Although not as pervasive in dust, diazinon had one of the highest 90<sup>th</sup> percentile concentrations in air (9.0 ng/m<sup>3</sup>). *Cis*- and *trans*-permethrin and PBO were also detected at relatively high levels in dust (7.04 µg/g, 16.5 µg/g, and 15.1 µg/g, respectively).

Table 2.12. Comparison of Indoor-air, Personal-air, and House-dust Levels in U.S.

Location	Season	Ranges and median and mean values					
		Chlorpyrifos			Diazinon		
		Range	Median	Mean	Range	Median	Mean
<b>Indoor air</b>		<b>Concentration (ng/m<sup>3</sup>)</b>					
Minneapolis, MN <sup>a</sup>	All	NL <sup>b</sup>	1.742	NL	NL	0.29	NL
NHEXAS Arizona <sup>c</sup>	All	<3.2-3280	8	NL	<2.1-20,500	4.6	NL
Brownsville, TX <sup>d</sup>	Spring	2.5-115	7.6	NL	1.6-60	1.4	NL
	Summer	5.7-67	24	NL	2.5-78	3.5	NL
Jacksonville, FL <sup>e</sup>	Summer	<2.5-2170	182	366.6	15-13,700	73	420.7
	Spring	NL	NL	205.4	NL	NL	109.2
Springfield, MA <sup>e</sup>	Winter	<2.5-1043	69	120.3	30-1080	21	85.7
	Spring	<4.5-252	<4.5	9.8	<22-1810	<22	48.4
	Winter	<3.5-291	<3.5	5.1	<28-28	<28	2.5
Raleigh, NC <sup>f</sup>	Fall	NL	NL	0.08	NA <sup>g</sup>	NA	NA
New Jersey area <sup>h</sup>	Fall	151.2-154.2	NL	NL	5.7	NL	NL
Cape Cod, MA <sup>i</sup>	All	<1-92	<1	NL	<1-550	<1	NL
<b>Personal air</b>		<b>Concentration (ng/m<sup>3</sup>)</b>					
Minneapolis, MN	All	NL	1.577	NL	NL	0.275	NL
NHEXAS Arizona	All	<19-175	NL	NL	<15	NL	NL
Jacksonville, FL	Summer	NL	NL	280.4	NL	NL	321.6
	Spring	NL	NL	182.8	NL	NL	112.7
	Winter	NL	NL	118.2	NL	NL	89
Springfield, MA	Spring	NL	NL	7.5	NL	NL	10.1
	Winter	NL	NL	5.9	NL	NL	1.4
<b>House dust</b>		<b>Concentration (μg/g)</b>					
Fresno, CA <sup>i</sup>	Fall <sup>j</sup>	0.2-33	NL	NL	0.7-169	NL	NL
	Fall <sup>k</sup>	<1	NL	NL	0.2-2.5	NL	NL
NHEXAS Arizona	All	<0.004-119	0.16	NL	<0.020-66	0.13	NL
Brownsville, TX	Spring	0.1-1.7	0.3	NL	0.1-1.8	0.06	NL
	Summer	0.2-1.7	0.56	NL	0.1-0.8	0.07	NL
Wenatchee, WA <sup>l</sup>	Spring <sup>j</sup>	<0.02-3.6	0.27	0.43	NA	NA	NA
	Spring <sup>k</sup>	<0.02-0.5	0.05	0.17	NA	NA	NA
Jacksonville, FL	Winter	NL	4.7	5.8	NL	0.4	1.7
Raleigh, NC	Fall	NA	NA	1.6	NA	NA	NA
New Jersey area <sup>h</sup>	Fall	0.53-15	NL	NL	0.08-0.74	NL	NL
PCS <sup>m</sup>	All	NL	0.308	0.607	NL	0.035	0.358
Cape Cod, MA <sup>i</sup>	All	<0.2-228	<0.2	NL	<0.2-51.0	<0.2	NL

Source: Modified from Gordon *et al.*, 1999.<sup>a</sup>Clayton *et al.*, 2003.<sup>b</sup>NL, not listed.<sup>c</sup>Gordon *et al.*, 1999.<sup>d</sup>Mukerjee *et al.*, 1997b.<sup>e</sup>Whitmore *et al.*, 1994.<sup>f</sup>Lewis *et al.*, 1994.<sup>g</sup>NA, not applicable.<sup>h</sup>Roinestad *et al.*, 1993.<sup>i</sup>Rudel *et al.*, 2003.<sup>j</sup>Bradman *et al.*, 1997.<sup>k</sup>Samples from agricultural family homes.<sup>l</sup>Samples from non-agricultural family homes.<sup>m</sup>Simcox *et al.*, 1995.<sup>n</sup>Whitmore *et al.*, 2003.



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### 2.3.6 Lead

Lead is a toxic metal that has been used in paints, gasoline, pipes, and ceramic glazes, and to a lesser extent, in caulk. Lead paint chips and soil contaminated with lead poses a major hazard to children when they ingest them. Over the long term, exposure to lead can lead to brain damage, decreased growth, hyperactivity, impaired hearing, and reproductive effects.

#### 2.3.6.1 *Health Effects of Lead*

The body mistakes lead for calcium and concentrates it in the bones, where it can leach into the blood. Both pre-natal and post-natal exposure, and exposure during childhood results in toxicity to the nervous system (Thacker *et al.*, 1992; Needleman *et al.*, 1979). Children, especially those younger than five, are more susceptible to lead than adults. Chronic, low-dose exposure to lead, often occurring in early childhood, can lead to decreased learning ability due to brain damage. Acute health effects, seldom seen, include seizures, paralysis, anemia, abdominal pain, constipation, vomiting, and decreased appetite.

Lead not only concentrates in bones, it also accumulates in the liver and kidneys. Lead has a half-life of 25 to 40 days in the blood and organs, and a half-life of 25 years in the bones. During pregnancy, lead mobilized from the bones and released into the bloodstream can pass through the umbilical cord and adversely affect the developing fetus. The blood lead level of concern for children, established by the U.S. Centers for Disease Control and Prevention in 1991, is 10 µg/dL (CDC, 1997). Significant decreases in IQ, behavioral problems, and learning impairment have been documented at blood-lead levels above this threshold. Recent research showing health effects at levels below 10 µg/dL now supports the growing consensus that there is no safe level for lead exposure.

#### 2.3.6.2 *Sources of Lead*

In the U.S., lead was eliminated from paints and gasoline in the late 1970s. Today, major sources of lead include old paint in homes built before 1978, lead pipes placed before the 1930s, lead solder in copper piping installed up to 1986, and soil by busy roads. Activities that disturb lead-based paint, such as remodeling or paint removal, can release large amounts of lead-bearing particles into the air, which may later settle in dust. Even without remodeling, deteriorating lead paint can accumulate in house dust. Often overlooked sources of lead include home remedies, cosmetics, hobby materials, and foreign-made, vinyl mini-blinds (ATSDR, 1999a; CPSC, 1996).

Next to resuspension of lead dust from lead-based paint, much of the lead present in indoor air appears to result from the infiltration of lead particles in outdoor air. Infiltrated and tracked-in lead dust, brought into the home from the workplace, can accumulate in carpets that can serve as a reservoir for lead-laden dust (U.S. EPA, 1997a). Research has shown that lead-dust loadings and concentrations per unit mass of dust are correlated with blood-lead levels, but no causal effect can be inferred from this association. Children's mouthing behaviors and activities that put them in direct contact with lead-contaminated surfaces increase their probability for exposure to lead by ingestion.

Candles containing a lead/metal core in the wick can emit lead into the air when burned. Van Alphen (1999) calculated the mean rate of lead release from seven candles was 770 µg/hour. In homes where candles are burnt once per week for several hours, lead air levels would be sufficient to drive the child's blood lead levels over the CDC level of concern of 10 µg/dL.

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### 2.3.6.3 Lead Concentrations

In a 1997 report, the U.S. EPA summarized findings from a literature review on the amount of lead in dust within carpets, furniture, and forced air ducts. A major drawback across studies was the inconsistency in the methods used to collect and analyze lead. In general, the average loadings are lower for furniture than for floors, window components, walls, and air ducts. Furniture, including upholstery and window treatments, has an averaging loading of less than 100 µg/ft<sup>2</sup>. The average loadings for forced air ducts exceed 100 µg/ft<sup>2</sup>, and can be as high as 1,000,000 µg/ft<sup>2</sup>.

Dust lead levels at schools are an important factor when determining children's total exposure to lead. In a recent comprehensive study of the environmental health conditions in California portable (relocatable) classrooms, ARB and DHS summarized floor-dust metal concentration data for 18 different metals (Whitmore *et al.*, 2003). All the elements, except copper, had higher dust loading in portable classrooms than traditional classrooms, though the differences were not statistically significant. As stated in the report: "Since the portable classrooms are generally newer, the lower concentration of lead may reflect the number of years' accumulation of the particles in the classroom." Table 2.13 reports the median and 95<sup>th</sup> percentile concentrations for lead in the floor dust of all classrooms sampled.

**Table 2.13. California Portable Classrooms Study Results for Lead in Floor Dust**

Element	Room Type	Concentrations (µg/g)		Loadings (ng/cm <sup>2</sup> )	
		Median	95 <sup>th</sup> Percentile	Median	95 <sup>th</sup> Percentile
Lead	All	61.6	189.5	6.6	58.4
	Portable	57.5	151.6	5.8	57.9
	Traditional	66.8	200.6	7.1	57.5

Between 1994 and 1998, DHS surveyed the lead content in paint and soil for a random sample of 200 California public elementary schools and child care facilities (DHS, 1998). Results were used to make predictions about the lead content in all of California's 5,000 schools. The study found: "As in California's housing, lead-containing paint is estimated to be in most California public elementary schools and child care facilities." DHS estimated that one-third of schools contain deteriorating lead paint, and nearly 6% have soil that is contaminated with lead exceeding U.S. EPA Lead in Soil Guidelines. The highest levels of lead in soil were found close to school buildings and near buildings built prior to 1940.

### 2.3.7 Mercury

There are three types of mercury: elemental (or metallic), inorganic (or mercury salts), and organic. In the past, inorganic mercury was added to skin-lightening creams and medicinal products. Today, compounds containing inorganic mercury are used as fungicides and antibacterials (ATSDR, 1999b). Metallic mercury can be found in thermometers, electric switches, fluorescent lights, thermostats, and other products. The primary source of organic mercury, specifically methylmercury, is fish and other types of seafood.

#### 2.3.7.1 Health Effects of Mercury

Workers exposed to mercury levels ranging from 25 to 60 µg/m<sup>3</sup> have experienced tremors, changes in personality, loss of sensation and muscle coordination, vision and hearing

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impairments, deficits in cognitive function, respiratory tract irritation, severe stomach comfort, skin rashes, and elevations in blood pressure and heart rate (OEHHA, 2000c). In combination with data from other published research, Carpi and Chen (2001) estimated that 10% of U.S. homes may have indoor mercury levels that exceed the U.S. EPA Reference Concentration of  $0.3 \mu\text{g}/\text{m}^3$ . Exceeding the U.S. EPA Reference Concentration may increase the likelihood of health impacts, but it might take years for the effects to be seen and they may not occur at all. Typical mercury exposures in indoor environments are not likely to produce developmental neurotoxicity.

### 2.3.7.2 Sources of Mercury

Indoor air is the second most common route of exposure to mercury in the general U.S. population (fish consumption is first) (NJ Mercury Task Force, 2002; Carpi and Chen, 2001; WHO, 1990). Mercury is an effective fungicide and bactericide, so it has been used as an intentional additive in many household products, including latex paints (interior use banned in 1990), contact lens solutions, and nasal sprays (ATSDR, 1999b). Mercury may also be found as an accidental contaminant in detergents and cleansers due to its extensive use in the chlor-alkali industry (U.S. EPA, 1999b). Elemental mercury's unique physical and chemical properties are the basis for its use in many household items. It expands uniformly with increases in temperature, does not cling to glass, and is liquid and can vaporize at room temperature. Mercury workers are exposed to greater levels of elemental mercury than the general population. Workers' families are also at increased risk; workers may bring home clothing and shoes contaminated with mercury in the workplace (ATSDR, 1999b). Elemental mercury is also used in some cultural practices, either carried as a good luck charm, or sprinkled in cars and homes or over infants (ATSDR, 1999b). With the removal of mercury in paint and the ban on the sale of mercury thermometers in California (SB633, 2002), exposures to mercury in indoor air in California should decrease over time. Table 2.14 lists the sources of mercury in homes.

**Table 2.14. Sources of Mercury in the Residential Environment**

Type of Mercury	Sources
Metallic	Thermometers, electric switches, fluorescent lights, thermostats, barometers, batteries, ethnic remedies, spiritual practices
Inorganic and organic	Latex paints, medicines, disinfectants, detergents, antiseptics

### 2.3.7.3 Mercury Concentrations

There is limited information on airborne exposures in the non-occupational environment. From June 2000 through March 2001, Carpi and Chen (2001) measured mercury concentrations in indoor air for nine residences and three businesses in the New York metropolitan area. Nearly all of the sites showed higher indoor levels than outdoor levels. The average indoor mercury concentration for all buildings was  $69 \text{ ng}/\text{m}^3$ , and was highly dependent on season.

Mercury is found in many household items, but unless the mercury-containing device is broken or disturbed, mercury does not pollute the air. Once released, however, mercury persists in the indoor environment for months or years after its first release, especially if the spill is not properly cleaned (ATSDR, 1999b). According to the New Jersey Mercury Task Force (2002): "Exposure to  $\text{Hg}^0$  (such as in certain cultural practices) can be significant with respect to health effects. As

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little as one drop (0.05 ml) of liquid  $\text{Hg}^0$  in a sealed bedroom-sized room (assuming a room volume of about  $33 \text{ m}^3$  and no air exchange) can result in an air concentration equal to the U.S. EPA Reference Concentration.”

Several nationwide incidences of non-occupational exposure to mercury have been documented. The Ohio Department of Health, in cooperation with the federal ATSDR, analyzed the indoor mercury concentrations in nine school gymnasiums (ATSDR, 2002). The highest detectable level of mercury in indoor air was  $1.6 \mu\text{g}/\text{m}^3$ . A *Morbidity and Mortality Weekly Report* summarized findings from the contamination of homes and schools with  $\text{Hg}^0$  in Palm Beach County, Florida (CDC, 1995). The report illustrated how quickly mercury can spread and how difficult it is to contain. The local authorities evacuated 50 homes, and closed schools until mercury levels dropped below  $10 \mu\text{g}/\text{m}^3$ . Seventeen homes contained vapor concentrations greater than  $15 \mu\text{g}/\text{m}^3$ . Table 2.15 shows the guideline levels for mercury.

**Table 2.15. Recommended Airborne Concentrations of Mercury**

Values	Mercury Vapor
ATSDR Minimum Risk Level (MRL)	$0.2 \mu\text{g}/\text{m}^3$
ATSDR recommended screening level	$1 \mu\text{g}/\text{m}^3$
ATSDR action level	$10 \mu\text{g}/\text{m}^3$
U.S. EPA reference concentration (RfC)	$0.3 \mu\text{g}/\text{m}^3$

### 2.3.8 Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) are a complex class of semi-volatile organic compounds and consist of two or more fused benzene rings. The larger PAH species are associated with fine or ultrafine particles; humans become exposed to a complex mixture of PAHs, not individual compounds (IARC, 1983; U.S. EPA, 1987c; Atkinson and Arey, 1989). Benzo(a)pyrene, a PAH with five fused benzene rings, is often considered to be the representative compound for PAHs (U.S. EPA, 1987c; IARC, 1983). PAHs are produced from incomplete combustion; indoor sources are primarily tobacco smoking, aerosols from hot cooking oil, woodburning, and infiltration of outdoor air.

#### 2.3.8.1 Health Effects of PAHs

Several individual PAHs are believed to be carcinogenic to humans. The U.S. Environmental Protection Agency (U.S. EPA) classifies benzo(a)pyrene as a possible human carcinogen; the International Agency for Research on Cancer (IARC) classifies benzo(a)pyrene as a probable human carcinogen (IARC, 1983; U.S. EPA, 1984). Other PAHs characterized by either IARC or U.S. EPA as possible human carcinogens include: chrysene, certain benzofluoranthenes, and indeno(1,2,3-cd)pyrene (IARC, 1983; U.S. EPA, 1984, ARB/OEHHA, 1994; ARB, 1994a). For most of the PAH species, there is currently insufficient evidence to classify these compounds as to their carcinogenicity.

#### 2.3.8.2 Sources of PAHs

Tobacco smoking, when present, was identified as the strongest indoor source of benzo(a)pyrene and other PAHs indoors in a study conducted in California (Sheldon *et al.*, 1993; Sheldon *et al.*, 1992b). Investigators have measured as many as 56 PAH species and derivatives in tobacco smoke; hundreds more may exist (Vu-Duc and Huynh, 1989; Wynder and Hoffman, 1964).

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Cooking activities were found to be the greatest contributor to PAH concentrations, second to infiltration of traffic -polluted outdoor air in a small study conducted by Dubowski *et al.* (1999). These investigators also determined candle- and incense burning contribute to indoor levels of PAHs. Siegman and Sattler (1996) determined that PAH concentrations in the fumes from hot cooking oil ranged from 1.08 to 22.8  $\mu\text{g}/\text{m}^3$ , compared to PAH concentrations found in a road tunnel with heavy traffic (2.6  $\mu\text{g}/\text{m}^3$ ) and an office with heavy cigarette smoking (1.2  $\mu\text{g}/\text{m}^3$ ). However, they caution that the oil droplets may not have the same carcinogenic potential as PAH from combustion aerosols.

Woodburning appliances contribute to indoor PAH levels. Fireplaces can emit soot and PAHs directly into the indoor air environment (Traynor *et al.*, 1987). While newer, more efficient airtight stoves appear to emit less than older, leakier woodstove models, poor maintenance and certain practices, such as operating the woodstove with the door open, can raise indoor air PAH levels substantially (Traynor *et al.*, 1987).

Transport of outdoor air into the home can introduce PAHs from outdoor sources such as traffic, diesel engines, power plants and agricultural burning (IARC, 1983; U.S. EPA, 1987c; Dubowski, 1999).

### 2.3.8.3 PAH Concentrations

The Air Resources Board funded two large studies to characterize indoor exposures to PAHs in California residences. In a northern California study, investigators measured PAHs inside 280 homes during the winter of 1992, evaluating the relationship between PAH levels and common indoor combustion sources (Sheldon *et al.*, 1993). Investigators reported average concentrations of 2.2  $\text{ng}/\text{m}^3$  of benzo(a)pyrene in smoker's homes, but only 0.83  $\text{ng}/\text{m}^3$  in nonsmoker's homes. Investigators reported that cigarette smoking significantly raised levels of 12 of 13 PAH species when compared to levels in nonsmokers' homes.

In the same study, fireplace and woodstove use raised average benzo(a)pyrene levels to about twice the levels found in homes with no obvious combustion sources. Researchers reported average benzo(a)pyrene concentrations of 1.2  $\text{ng}/\text{m}^3$  compared to 0.83  $\text{ng}/\text{m}^3$  measured in "no source" homes. The benzo(a)pyrene level of 0.83  $\text{ng}/\text{m}^3$  was primarily attributed to infiltration of smoke from community woodburning, a value well above the typical average outdoor levels in California (Atkinson and Arey, 1989).

The second ARB-funded study was conducted in Southern California homes. In this study, Sheldon *et al.* (1992b) found polluted outdoor air to be a major source of indoor PAHs when other combustion sources were absent in the home. Other investigators have reported similar findings (Waldman *et al.*, 1989; Lioy and Greenberg, 1990; Naumova *et al.*, 2002). In two Los Angeles area communities, Naumova *et al.* (2002) measured PAHs in homes located near busy traffic areas and reported significantly lower levels of particle-bound PAHs indoors than outdoors. Concentrations were dominated by coronene, an indicator of motor vehicle-related pollution. Investigators stated that these data suggested that indoor concentrations were driven by outdoor PAH pollutant levels. Cigarette smoking was also a major contributor to indoor PAH levels in the study of southern California homes (Sheldon *et al.*, 1992b). In smokers' homes, investigators reported statistically significant increased concentrations relative to nonsmoking homes of nine PAH species, including benzo(a)pyrene during the daytime, and six PAH species during the night.

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Investigators in other states have reported substantial increases in benzo(a)pyrene indoors as a result of tobacco smoking in the home (Kanarek *et al.*, 1985; Chuang *et al.*, 1988; Turk *et al.*, 1987). In a study of PAH levels in 15 public buildings in the Pacific Northwest, Turk (1987) reported average levels of benzo(a)pyrene of 1.07 ng/m<sup>3</sup> smoking areas compared to 0.39 ng/m<sup>3</sup> in nonsmoking areas. The mean indoor-to-outdoor ratio was 7.6.

### 2.3.9 Radon

Radon is a naturally-occurring radioactive gas derived from the decay of radium-226 (a decay product of uranium-238) present in small amounts in some soils, rocks, and water. Radon itself is relatively harmless, but its decay products, called radon daughters or radon progeny, can accumulate in the lung and cause cancer. A statewide survey (Liu *et al.*, 1991) indicates that only 0.8% of California residences have annual radon levels above 4.0 pCi/l, U.S. EPA's recommended mitigation level, and the statewide radon average is about 1 pCi/l. However, due to the potency of radon daughters, a preliminary estimate based on an extrapolation from national data indicates that radon contributes to about 1500 excess cancer deaths per year in California. There is substantial uncertainty surrounding this estimate, however, in part due to its inseparability from the risk from exposure to tobacco smoke, and as explained further below.

#### 2.3.9.1 Health Effects of Radon

Causal associations between exposure to radon and its progeny and lung cancer have been demonstrated in many epidemiological studies of underground miners. The National Research Council (NRC) Committee on the Biological Effects of Ionizing Radiations has updated estimates of radon potency and lung cancer deaths over a number of years, at times increasing or decreasing the estimated potency or deaths (NRC, 1988; NRC, 1990; NRC, 1999a). In the most recent assessment, NRC (1999a) estimated excess lung cancer deaths in the U.S. due to residential radon exposure using two different models. The results range from 15,400 to 21,800 deaths, for the age-duration and age-concentration risk models, respectively. The U.S. EPA (2003c) modified the NRC models and reported 21,000 excess lung cancer deaths (90% confidence interval or CI: 9,000 to 50,000) due to residential radon exposure nationally. Concurrent exposure to radon and smoking show synergistic effects, that is, the risk of lung cancer is higher than predicted by adding the individual risks (NRC, 1999a). It is difficult, if not impossible, to separate lung cancer risk from ETS and that from radon (NRC, 1999a).

To provide a rough estimate of the risk from radon in California for the purposes of this report, the first-order estimation of radon-induced lung cancer deaths in California was derived using simple linear extrapolation by population number, smoking prevalence, and average indoor radon concentrations. The national estimates (21,000 deaths/year) were multiplied by the fraction of the U.S. population in California (34 million out of 281 million, or 12%; U.S. Census 2000 data); the ratio of average radon concentrations (CA: 1 pCi/l; U.S.: 1.25 pCi/l; ratio of 0.8); and the smoking prevalence in California (CA: 16.4%; U.S.: 22.5%, ratio of 0.73; CDC, 2004). This preliminary calculation yields an estimate of about 1,500 (90% CI 600-2,100) excess cancer deaths per year in California attributable to radon exposure.

However, this estimate is likely to be an over-estimate, for several reasons. First, a recent study in the Sierra foothills region found that the percentage of households exceeding 4 pCi/l is far less than the percentage found for that region in the previous statewide survey (3% vs. 25%), so the actual statewide radon average may be notably lower than the previous estimate of 1 pCi/l (Tsai and Waldman, 2005). Additionally, current indoor radon levels may be lower than the measurements taken 15 years ago because new construction may be more radon resistant.

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These factors would lead to a lower statewide radon average; thus the risk estimate presented here may be an overestimate, and should be viewed as preliminary. Finally, as discussed in an earlier section, smoking rates and exposure to secondhand smoke have decreased substantially in California as a result of state regulations and public education efforts. This may reduce the opportunity for synergistic effects of radon and tobacco smoke to occur, resulting in fewer cases of cancer.

Although the toxicity of radon is relatively certain, based on strong epidemiological studies, many uncertainties associated with the estimation of risk in California remain to be explored. Detailed information for region-specific residential radon exposures, in combination with demographic data (e.g., age, gender) and smoking status (current and previous smoking; non-smoking) would be required to perform a more accurate radon risk assessment for California, but such an effort is beyond the scope of this report. Additionally, a more refined estimate may not be useful, for several reasons. A statewide solution in a state with such low average levels of radon is not useful for addressing radon. The most effective approach to reducing risk from radon is to avoid tobacco smoke (Mendez *et al.*, 1998; NRC, 1999a), and California has aggressive state programs to promote reductions in smoking. Local building codes to prevent radon intrusion in regions with elevated radon are another preventive measure that could provide some protection. However, following the simplified estimation approach above, even if the statewide indoor radon average were reduced to the ambient level, 0.5 pCi/l, about 750 (300-1,050) lung cancer deaths would still be expected to occur per year due to natural radiation.

While the majority of radon risk is associated with inhalation exposure from air, naturally occurring radon in water also poses a risk. About 168 cancer deaths per year are estimated to occur in the U.S. from radon in drinking water – 89% from lung cancer caused by breathing radon released from water (e.g., showering or flushing the toilet), and 11% from stomach cancer caused by drinking radon-containing water (NRC, 1999b).

### 2.3.9.2 Sources of Radon

Indoor radon derives from various sources in the vicinity of a building: (a) release from soils underneath the building (generally the most important source), (b) building materials enriched with uranium-238 (e.g., granite or concrete blocks used in the foundation or walls), (c) use of radon-enriched local well water (especially for bathing and appliance use), and (d) outdoor air. Elevated indoor radon levels are generally due to the combination of a rich source, a driving force into the building, and a relatively low dilution rate for fresh air (Eichholz, 1987).

### 2.3.9.3 Radon Concentrations

Samplers are available to test indoor radon levels at reasonable cost. Measurement techniques include charcoal canisters, alpha track detectors, electret ion chamber detectors or scintillation flasks. Radon concentrations are reported as the number of radioactive decays per time in a volume of either air or water, or picoCuries per liter (pCi/l). The U.S. EPA has issued recommendations to reduce the health risks from radon exposure in homes. Immediate corrective action should be pursued when indoor air radon levels are above 200 pCi/l. For radon levels between 20 and 200 pCi/l, corrective action should be pursued within a few months. For radon levels between 4 and 20 pCi/l, corrective action should be pursued within a few years (U.S. EPA, 2002a).

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A statewide survey in California found that only 0.8% of California residences have annual radon concentrations exceeding 4 pCi/l (Liu *et al.*, 1991). This study examined annual average radon concentrations in over 300 homes in a population-based survey and determined that the average radon level in California homes is 1 pCi/l. Areas with higher than average radon levels include the Central Valley, the Sierra Nevada mountains and foothills, the valleys east of the Sierra Nevada, and Ventura and Santa Barbara counties. Surveys of California public elementary schools demonstrated similar geographic patterns for areas with elevated indoor radon (Churchill and Youngs, 1993; Zhou *et al.*, 1998).

The National Residential Radon Survey was conducted by the U.S. EPA in 1989-90 to assess annual average radon concentrations in U.S. residences (Marcinowski *et al.*, 1994). The survey estimated an arithmetic annual average radon concentration in U.S. homes of  $1.25 \pm 0.12$  pCi/l, and about 6.0% of homes had radon levels greater than the U.S. EPA action level of 4 pCi/l.

Homes with elevated levels of radon are more commonly found in geographical areas with higher natural levels of soil radioactivity (i.e., uranium). However, predicting residential radon concentrations is difficult due to factors associated with home construction and operation (Churchill, 1997). Nonetheless, building factors, such as ventilated crawl space, and age of the buildings may affect the indoor radon (Tsai and Waldman, 2005).

### 2.3.9.4 Activities and Policies to Address Radon in California

The Department of Health Services' (DHS) Environmental Management Branch, within the Division of Drinking Water & Environmental Management, manages the state's Radon Program. The Radon Program goals are to increase the public's awareness of the risks of radon in indoor air and reduce exposure by encouraging mitigation in buildings with elevated levels of radon. The program (ongoing for 14 years) promotes radon testing of homes, businesses, and schools through outreach programs and distribution of free radon test kits; maintains a statewide database; and is responsible for listing individuals certified to perform radon services in California. DHS encourages all homeowners to test their home for radon, and provides test and remediation information to the public.

In addition, yearly intensive radon screening studies of selected counties identify areas with the potential for elevated indoor levels of radon based on short-term indoor radon measurements. Selection of counties and sampling sites is based primarily on geology because uranium occurrence is not random: it is largely influenced by geology. There are three primary benefits arising from the screening studies: 1) outreach and public education: approximately 25,000 recruitment/radon information letters are sent to residents of each selected county; 2) identification of areas of high indoor radon potential: mitigation and construction of radon resistant homes are encouraged in these areas; and 3) production of detailed radon potential maps. To date, three such maps have been produced (Santa Barbara, Ventura and, soon to be released, Los Angeles) and are available through the Program's web page at [www.dhs.ca.gov/radon](http://www.dhs.ca.gov/radon) and distributed to interested city and county agencies.

### 2.3.10 Asbestos

Asbestos is a naturally occurring mineral fiber with strong commercial appeal due to its physical properties. It is a poor conductor, but a good insulator, and is strong, flexible, non-corrosive, and flame-resistant. Asbestos is a commercial term for a number of naturally-occurring minerals: chrysotile belongs to the serpentine group, while amosite, crocidolite, tremolite, actinolite, and anthophyllite are in a group called amphiboles. When inhaled, asbestos fibers penetrate deep



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into lung tissues where they cannot be expelled or destroyed by the body. Federal and International agencies recognize that asbestos is a carcinogen. In 1986, asbestos was identified as a toxic air contaminant under California's Toxic Air Contaminants Program (AB 1807). Exposure to asbestos is associated with increases in non-malignant respiratory symptoms, and may cause asbestosis, lung cancer, and mesothelioma (ATSDR, 2001). Indoor asbestos is primarily found in older homes in pipe and furnace insulation, shingles, millboard, textured paints and other coating materials, and floor tiles. Due to the success of remediation efforts, asbestos concentrations are generally quite low in most buildings today.

### 2.3.10.1 *Health Effects of Asbestos*

Asbestos fibers are so small and thin that they can penetrate to the small alveoli in the lungs. Once the fibers are inside the lungs, the body's defense mechanisms attempt to break them down and remove them. Despite these attempts, many fibers remain in the body and are potential disease-causing agents. Each fiber is treated as a foreign body; inflammations develop as the body tries to neutralize, break down or remove the sharp, irritating fibers. Fibers longer than 5  $\mu\text{m}$  in length tend to have the greatest health impact. These fibers and the bodies defense mechanisms lead to the development of the various kinds of asbestos-caused diseases.

Lung cancer is the predominant asbestos-related disease, accounting for the majority of deaths from asbestos exposure. Asbestos workers who smoke are at greater risk of developing lung cancer than workers who do not smoke. Asbestosis, a diffuse fibrous scarring of the lungs characterized by shortness of breath, mainly arises after long-term heavy exposure to asbestos. Mesothelioma, a rare cancer of the lining of the chest and abdomen, does not appear to be influenced by smoking. A study of vermiculite miners in Libby, Montana revealed a 4.2% death rate due to mesothelioma (McDonald *et al.*, 2004). Based on the high-end estimate of the potency of mixed asbestos fibers, breathing air that contains 100 fibers/ $\text{m}^3$  (0.0001 fibers/ml) poses a one to two in ten thousand excess lifetime risk of cancer (ARB, 1986). Others (U.S. EPA, 1994) have made similar estimates.

Non-occupational exposures are generally low compared to occupational exposures. Workers' families have developed asbestos-related pulmonary disease (Whitehouse, 2000; Peipins *et al.*, 2003), lung cancer and mesothelioma (Magnani *et al.*, 2001) from para-occupational exposure to asbestos, such as inhalation of asbestos fibers released by clothing and equipment brought home from job sites. People living in the vicinity of asbestos mines and factories may be at risk from neighborhood exposure to asbestos. Although the risk to the general population is minimal, no safe exposure level for asbestos has been established.

### 2.3.10.2 *Sources of Asbestos*

For 30 years following World War II, asbestos was extensively used in the renovation and construction of homes, schools, and public buildings. Asbestos was once found in nearly 3,000 different types of commercial products, including older plastics, paper products, brake linings, floor tiles, textiles, sealants, cement pipe, cement sheets, and insulation (NTP, 2002). It is now prohibited in the manufacture, processing, and importation of most products by the 1989 Asbestos Ban and Phase Out Rule (40 CFR 763 Subpart I, Sec. 762.160 - 763.179). Some asbestos paper products and asbestos-cement products remain in use today.

Friable asbestos-containing material (ACM) can be crumbled by hand pressure when dry, releasing fibers to air for potential exposure by individuals. Non-friable ACM can become friable

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during abatement and maintenance activities, and with age. The three most common asbestos forms used in construction are amosite, crocidolite, and chrysotile (DHS, 2000). When the minerals are crushed, they break into tiny fibers invisible to the naked eye, but distinguishable under light microscope. Chrysotile fragments into curly fibers; the other types release tiny, needle-like fibers.

Homes built in areas of naturally occurring asbestos may become contaminated with tracked-in asbestos and infiltration of fibers that are suspended in outdoor air. Once such fibers are indoors, they can be resuspended by normal household activities, such as vacuuming (OEHHA, 2000b).

### 2.3.10.3 Asbestos Concentrations

For the purposes of counting asbestos fibers in samples, regulatory agencies commonly count as fibers those particles of asbestos minerals at least 5  $\mu\text{m}$  in length and with length:width ratios of 3:1. For other purposes, such as detecting fibers in bulk building materials, asbestos particles with length:width ratios of 5:1 are counted.

Asbestos concentrations in most buildings are quite low. Crump and Farrar (1989) examined indoor asbestos concentrations for 49 public buildings located in six cities across the U.S. The concentration of all fibers was 0.00073 f/ml, and for fibers greater than or equal to 5  $\mu\text{m}$ , the average concentration was 0.00007 f/ml. The mean indoor concentration was 0.00020 f/ml for buildings with no known asbestos, 0.00059 f/ml for buildings with asbestos in good condition, and 0.00073 f/ml for buildings with damaged asbestos. There were no statistically significant differences in airborne asbestos levels among the three categories of buildings. For the 43 buildings that contained ACM, the average indoor levels for fibers greater than or equal to 5  $\mu\text{m}$  were 0.00005 f/ml; these levels were indistinguishable from outdoor levels (Lee *et al.*, 1992).

In a nationwide study conducted by Lee *et al.* (1992), airborne asbestos concentrations were made for 315 public, commercial, residential, school, and university buildings. The mean indoor level was 0.02485 f/ml for total asbestos structures and 0.00013 f/ml for fibers longer than 5  $\mu\text{m}$ . Indoor total asbestos was significantly higher than outdoor total asbestos for all building types. In comparing indoor and outdoor levels of fibers greater than or equal to 5  $\mu\text{m}$  for building types, indoor levels were higher than outdoor levels only for schools ( $p=0.003$ ). Only 52% of all indoor samples contained asbestos fibers. Approximately 92% of fibers were shorter than 5  $\mu\text{m}$  in all buildings, and 2% of fibers were amphiboles. 90% of the samples for fibers longer than 5  $\mu\text{m}$  had levels less than 0.00071 f/ml and 0.00054 f/ml in schools and public buildings, respectively.

Corn *et al.* (1991) reported asbestos concentrations for a portion of the study conducted by Lee *et al.* (1992) involving 71 occupied schools in eight different states. Schools from different states did not differ statistically in their levels of airborne asbestos. The type of asbestos-containing materials (ACM) present (i.e., chrysotile or amphibole) and the condition of ACM were not significantly correlated with levels of indoor asbestos. Most of the asbestos fibers were chrysotile (95%) with an average of 0.018 f/ml, and few were longer than 5  $\mu\text{m}$  (7.9%). The mean indoor concentration of fibers over 5  $\mu\text{m}$  long was 0.00024 f/ml. Total asbestos structures found indoors averaged 0.20 f/ml.

### 2.3.11 Polybrominated Diphenyl Ethers and Other Endocrine Disrupters

Endocrine disrupters are substances that alter the normal function(s) of the endocrine systems of animals and humans and adversely affect growth, development or reproduction. They can act

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like a natural hormone, bind to a receptor and prevent a normal response, or interfere with the way natural hormones and receptors are synthesized or controlled. Public attention has been drawn to endocrine disrupters that mimic or block the natural effects of female sex hormones (estrogens), but they can also affect male sex hormones, development and behavior. The range of substances that cause endocrine disruption is wide and varied, and includes both natural and synthetic chemicals.

Polybrominated diphenyl ethers (PBDE), a chemical class of endocrine disrupters, are added to plastics and textiles to meet fire safety regulations. The PBDE class is comprised of 209 possible congeners, differentiated by the position and number of bromine atoms on the two phenyl rings. U.S. EPA has classified deca-BDE as a possible human carcinogen since it has been linked to development of liver tumors in rats and mice. PBDEs have been identified as HAPs and California (TACs).

Phthalates are another group of chemicals that have been investigated for their potential endocrine-disrupting activities and many members of this group of chemicals, e.g., di-butyl phthalate (DBP), di-(2ethylhexyl) phthalate (DEHP), have been implicated as endocrine disrupters based on sufficient evidence in laboratory animals. Many are used to provide flexibility to plastic products and have been used in the manufacture of many products including toys, vinyl upholstery, shower curtains, raincoats, garden hoses, surgical gloves, medical tubing, and blood storage bags.

### 2.3.11.1 *Health Effects of PBDEs*

Research has shown that PBDE compounds are toxic and bioaccumulate similarly to polychlorinated biphenyls (PCBs) and dioxins and have genotoxicity profiles similar to PCBs. PBDEs have similar chemical structures to PCBs and the human thyroid hormone, thyroxine (T4). Toxicological endpoints of PBDEs are suspected to be thyroid hormone disruption, neurodevelopmental deficits, and cancer (McDonald, 2002).

Neurodevelopmental deficits have been linked to PBDE exposure (Eriksson *et al.*, 1998, 1999; Viberg *et al.*, 2000, 2001). Other results indicated deficits in brain development and possibly changes in the cholinergic system. Changes in the cholinergic system interfere with choline acetyltransferase activity and contribute to motor and mental impairments in animals. A no-observable-effects level for neurobehavioral effects has not been established for PBDEs (McDonald, 2002).

Evidence shows that exposure to PBDEs can lead to disruption of endocrine function in a number of wildlife species. Effects suggesting endocrine disruption have been reported in mollusks, crustaceans, fish, reptiles, birds, and mammals in various parts of the world. There is limited evidence in humans that adverse endocrine-mediated effects have resulted from either intentional or accidental exposure to chemicals. Many of these chemicals, even at relatively low levels, are known to affect growth, reproduction, and development of organisms in the ecosystems. The impact of these substances on human health is still under investigation.

### 2.3.11.2 *Sources of PBDE*

PBDEs are added to plastics and textiles to meet fire safety regulations. The production of PBDEs has steadily increased since the 1970s. Commercial forms of PBDEs include deca-, octa- and penta-BDE. In 1999, its use in the United States was estimated at 25,000 tons or 44%

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of the total global use (Hale *et al.*, 2002). California is a major user of fire retardants due to strict regulations concerning consumer safety.

Commercial penta-BDE is added to soft polyurethane foam cushions, upholstery textiles and mattresses. Commercial octa-BDE is incorporated into plastics used for hard casings of office equipment, fax machines, computers, telephone handsets and car trim. Commercial deca-BDE is utilized in high-impact plastics found in televisions, computers, stereos and other electronic equipment.

Historically, di-2-ethylhexylphthalate has constituted approximately 50% of all the phthalate ester plasticizers used. In 1994, production of DEHP was approximately 258 million pounds (IARC, 2000). DEHP is the single largest volume member of the di-octyl phthalates. However, in recent years the use of DEHP has declined because of health concerns. It is no longer used in plastic food packaging or baby teething rings. Many toy manufacturers have discontinued its use in toys, and it is being replaced by linear phthalates and other plastomers in other applications (ATSD, 2000).

### 2.3.11.3 PBDE Concentrations

The air and dust inside U.S. homes are likely to contain a wide variety of chemicals, many of which are identified as endocrine disrupting substances. A recent study (Rudel *et al.*, 2003) found numerous endocrine disrupting compounds in indoor air and dust obtained from 120 homes in Cape Cod, Massachusetts. The most abundant compounds in air included bis(2-ethylhexyl)phthalate (DEHP), (median=77 ng/m<sup>3</sup>, max= 1000 ng/m<sup>3</sup>); o-phenylphenol (disinfectant) (median=70 ng/m<sup>3</sup>, max=590 ng/m<sup>3</sup>); 4-nonylphenol (detergent metabolite) (median=110 ng/m<sup>3</sup>, max=420 ng/m<sup>3</sup>); and 4-*tert*-butylphenol (adhesive) (median=16 ng/m<sup>3</sup>, max=290 ng/m<sup>3</sup>). The penta- and tetrabrominated diphenyl ethers used as flame retardants were frequently detected in dust. Numerous pesticides were detected in air and dusts from these homes, the most abundant being permethrins and the synergist piperonyl butoxide. The banned pesticides heptachlor, chlordane, methoxychlor, and DDT were also frequently detected. The median concentration of DEHP in the homes studied was 590 ng/m<sup>3</sup> in the air and 4.98 µg per gram of dust collected. The authors noted that “detected concentrations exceeded government health-based guidelines for 15 compounds, but no guidelines are available for 28 compounds, and existing guidelines do not consider endocrine effects.”

PBDE congeners were quantified in samples of human breast milk collected in Sweden during the period from 1972 to 1997 (Noren and Meironyte, 1998). This Swedish study reported that levels of PBDEs in breast milk had increased 40 fold since 1972, indicating a doubling every five years. In a 2003 U.S. study, levels of PBDEs in women's breast milk measured in Texas were 10 to 100 times higher compared to levels found in Europe (Schechter *et al.*, 2003). Two recent U.S. studies in California (Petreas *et al.*, 2003) and Indiana (Mazdai *et al.*, 2003) also report similar results with levels from 10 to 100 times higher than levels reported in Europe (Schechter *et al.*, 2003).

PBDEs have been found in a wide variety of environmental samples, including fish, birds, soil sediments, air, marine mammals, and human blood (Strandberg *et al.*, 2001, Darnerud *et al.*, 2001). Penta-BDE has been detected among 89% of the fish collected from two large Virginia watersheds (Hale *et al.*, 2001a).

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An air study found that PBDE compounds were widely distributed and that PBDEs with lower molecular weight, such as penta-BDE (which is found mostly in the vapor phase), can be transported through the atmosphere to remote areas (Strandberg *et al.*, 2001).

### 2.3.12 Non-Industrial Workplace Exposure to Air Pollutants

Non-industrial workplaces provide unique situations for exposure to indoor air pollutants. Products and activities associated with non-industrial workplaces such as beauty salons, hospitals, dry cleaners, medical laboratories, retail shops, copy shops, and other workplaces can lead to elevated levels of air pollutants. Despite regulations for pollutant levels and ventilation requirements, some workers in these environments experience adverse health effects related to indoor environmental quality.

Under CCR Title 8, Section 5155, the California Occupational Safety and Health Standards Board sets permissible exposure limits (PELs) and other requirements to limit employee exposure to airborne contaminants. The PELs legally apply to both industrial and non-industrial workplaces. The Cal/OSHA PELs include 8-hour exposure limits designed to protect healthy working adults, as well as short-term and ceiling exposure limits when they are necessary to prevent acute effects. However, they are not sufficient to prevent all health impacts for all working individuals, and they do not address possible impacts on those with asthma or reactions by sensitive individuals to low levels of chemicals. Cal/OSHA's Indoor Air Quality Policy and Procedure (C-48) states: "Most complaints about the quality of indoor air arise from employees who work in non-industrial environment. Approaches using traditional industrial hygiene techniques usually demonstrate compliance with 8 CCR section 5155 (PELs) despite the persistence of IAQ complaints from the building occupants."

Cal/OSHA also has a regulation designed to provide adequate ventilation while workers are at work. Title 8, Section 5142, *Mechanically Driven Heating, Ventilating and Air Conditions (HVAC) Systems to Provide Minimum Building Ventilation*, requires HVAC systems to be operated during hours of occupancy to provide at least the amount of outside air required by the State Building Code at the time of permitting. It also contains maintenance and record-keeping requirements.

#### Cal/OSHA data on workplace investigations

A review of Cal/OSHA inspection data indicates ventilation requirements are often not met. From 1995 through 2001 data show that citations for violations of Title 8, Section 5142 were issued during 514 on-site inspections (Gold, 2005). Of these citations, the majority (260) were in services, with 114 in schools and 33 in health care. Citations for violations of Section 5142 were issued in 112 inspections in Public Administration.

Indoor air quality problems in non-industrial workplaces range beyond inadequate ventilation. A review of data collected by Cal/OSHA from 1997 through 1999 reveals that 849 cases were coded as being related to poor indoor air quality (Gold, 2005). However, because all inspections that included IAQ concerns were not necessarily coded as IAQ, the actual number of inspections involving indoor air issues may have been more than twice this number (Gold, 2005). Of the 849 cases, 353 inspections were in service workplaces and 128 were in public administration (typically offices). These numbers indicate that problems exist with indoor air quality in these locales, but information on specific pollutants or conditions leading to the inspections and possible citations are not readily available.

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In addition to the indoor air quality problems documented in Cal/OSHA records, the Hazard Evaluation Section and Information Service (HESIS) within DHS receives many inquiries from individuals who are experiencing illnesses associated with poor indoor air quality (Katz, 2005). Problems are largely associated with several types of personal-services and community-services environments. Typically, the individuals affected in these environments include employees, clients or customers, students, patients, and self-employed workers. The following discussion includes some non-office, non-industrial indoor environments with widespread indoor air problems documented by the Occupational Health Branch of DHS, the National Institute of Occupational Safety and Health (NIOSH) within CDC, and other scientific investigators.

### Schools, preschools, nursery schools, daycare

Teachers and other school staff frequently report illnesses associated with poor indoor air quality. Often the problems contributing to those illnesses are not adequately remedied by school administrators for months or even years. At the request of the Legislature, ARB and DHS (2004) conducted a study of the environmental conditions in both portable and traditional classrooms throughout California. They found that a number of indoor air quality problems were widespread in California. The most serious indoor air quality problems included:

- Inadequate ventilation with outdoor air during 40% of classroom hours, and seriously deficient ventilation 10% of the time, largely due to teachers turning off the ventilation systems due to excess noise produced by the systems.
- Formaldehyde concentrations above guidelines for preventing acute eye, nose, and throat irritation in 4% of the classrooms, and exceeding guidelines for preventing long-term health effects, including cancer, in all classrooms.
- Obvious mold in about 3% of classrooms, and water stains, excess wall moisture, and other potential mold indicators in about one-third of classrooms.

Other studies have identified air quality problems in classrooms as well:

- Defresne *et al.*, (2002) found that students in a 3-hour biology lab were exposed to average formaldehyde levels of 0.25 mg/m<sup>3</sup> (200 ppb) in one classroom, and 0.632 mg/m<sup>3</sup> (510 ppb) in another classroom. These values are well above the OEHHA 8-hour guideline level of 27 ppb.
- In another study (Ryan *et al.*, 2002), investigators measured personal exposures of art students to VOCs in a university art building. Concentrations of methylene chloride, a carcinogen, were elevated near the print cleaning operation, at 27.2 µg/m<sup>3</sup> (std. dev. 48 µg/m<sup>3</sup>). Concentrations were well below existing occupational limits for 40-hour exposures, but the elevated area exceeded the Proposition 65 no significant cancer risk level of 10 µg/m<sup>3</sup>.

### Photocopy centers

Numerous VOCs are emitted during the photocopy process (see Section 2.3.2.2). Stefaniak *et al.*, (2000) measured VOC concentrations in three copy centers on a university campus. 38 VOCs were detected in the personal air samples, with toluene being the highest at 690 ppb. The maximum area air concentration measured was 1,132 ppb toluene. The time-weighted average personal exposures to VOCs were 100 times less than OSHA PELs. However, the toluene levels exceed OEHHA's chronic REL of 75 ppb, a guideline designed to protect against the effects of long-term exposure, and exceed the California Proposition 65 warning level of 172 ppb (650 µg/m<sup>3</sup>) for reproductive toxicity. Real-time total VOC (TVOC) concentrations ranged from < 71 to 21,300 ppb. The authors conclude that "even though concentrations of individual VOCs are well below regulatory limits, the time-weighted average TVOC concentration can be well above suggested levels known to cause perceived and physiological health effects in controlled laboratory studies".

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### Indoor sporting events

Recreational events are attended by a wide cross section of the population, including individuals with heart conditions who are more susceptible to the effects of carbon monoxide (CO). Indoor CO levels can exceed the PEL of 25 ppm at indoor sporting events such as motocross competitions, tractor pulls, monster truck shows, and automobile demolition derbies, where internal combustion engines are operated in indoor environments (Levesque *et al.*, 2000; Levesque *et al.*, 1997; MMWR, 1994). Monster truck shows and tractor pulls in Cincinnati, Ohio during 1992-1993 were found to have average levels of 79-140 ppm CO during events compared to pre-event average levels of 13-23 ppm, and a peak observation of 283 ppm (Boudreau *et al.*, 1994). The authors noted that the observed concentrations varied inversely with the arena seating level (lower levels with greater height of seats). Similar CO concentrations were observed by Levesque *et al.* (2000) during monster truck shows and demolition derbies in Canada, and in motocross events (Levesque *et al.*, 1997).

Elevated CO and nitrogen dioxide (NO<sub>2</sub>) concentrations are often present in indoor ice-skating rinks. Pelham *et al.* (2002) found that, in 33 investigations of indoor skating rinks, the majority of the reviewed studies reported CO levels in excess of 25 ppm, and several studies reported NO<sub>2</sub> concentrations in excess of 1 ppm, well above occupational levels (STEL = 1 ppm) and well above the one-hour state ambient air quality standard of 0.25 ppm. The two most identified causes for high CO and NO<sub>2</sub> levels were the ice surface refinisher (Zamboni) and inadequate ventilation systems.

### Hair salons and nail salons

Hair and nail products typically contain toxic and irritating chemicals, though air concentrations tend to be well within the applicable occupational standards (Labreche *et al.*, 2003; Leino *et al.*, 1999; NIOSH, 1992). Many are highly volatile or are sprayed as aerosols, yet there is often inadequate ventilation to remove the airborne chemicals. Many salons are leased facilities where the business owner has little or no control over the ventilation system, which may distribute the pollutants to adjoining businesses. HESIS has received numerous inquiries from cosmetologists and cosmetology students about this problem. Health concerns include asthma and other respiratory problems, dizziness with headache, and potential effects on pregnancy. Because of the high number of complaints and the seriousness of the health concerns, HESIS has conducted several field investigations in this field of personal-service businesses (HESIS, 2004).

Hiipakka and Samimi (1987) measured several VOCs in personal air samples of nail salon operators. Reported values were below OSHA PELs; however, workers reported health symptoms. The incidence of throat irritation was significantly elevated relative to a control group. Nail sculptors consistently reported more nose and skin irritation, drowsiness, dizzy spells, and trembling of the hands than the control group. The mean time-weighted average concentration of chemicals reported were: ethyl methacrylate, 4.5 ppb; isopropyl alcohol, 15.6 ppb; butyl acetate, 0.4 ppb; toluene, 0.8 ppb; and polymethacrylate dust, 0.9 mg/m<sup>3</sup> for respirable dust.

### Other non-industrial workplaces with unique indoor exposure scenarios

Workers in many types of workplaces and businesses are exposed to air pollutants associated with the type of work they perform. Large air quality studies of these businesses are lacking; however various small studies, complaint records, and investigations have shown that there are indoor air quality problems in many other types of non-industrial workplaces as well. Some of these workplaces or services include:

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**Dry Cleaners** - Residual solvents used in dry cleaning volatilize off the clean clothes leading to elevated levels in dry cleaner shops. Perchloroethylene has been the mostly widely used dry cleaning solvent; however other solvents (ethylbenzene, *m,p*-xylene, and *o*-xylene) have been used to some extent. In studies in New York, concentrations of perchloroethylene in 13 apartments connected to or near dry-cleaning shops ranged from 7.6 to 23,000  $\mu\text{g}/\text{m}^3$ , averaged 5000  $\mu\text{g}/\text{m}^3$ , and showed a median level of 1400  $\mu\text{g}/\text{m}^3$ , well above the New York Department of Health guideline of 100  $\mu\text{g}/\text{m}^3$  (NY DOH, 2003) and the OEHHA chronic REL of 35  $\mu\text{g}/\text{m}^3$  and any acceptable cancer risk levels.

**Janitors** - Occupational asthma and other respiratory problems can be caused or aggravated by chemical cleaning products used inside schools, hotels, medical settings and other service facilities. California has nearly 400,000 people who regularly work with cleaning products. The Occupational Health Branch (OHB) of DHS found that the rate of work-related asthma in janitors is nearly double the rate in the overall workforce. Bystanders in health care and other industries are often indirectly exposed to cleaning chemicals as well. Nurses and nursing aides constituted 20% of the bystanders who developed asthma in the OHB study (DHS, 2004b; DHS, 2004c).

**Hospitals, jails, etc.-** Tuberculosis, a bacteria transmitted by airborne particles, is a substantial risk for persons in group-living environments. These environments include hospitals, prisons, jails, homeless shelters, drug treatment clinics, residential facilities for HIV-infected persons (e.g., hospices or group housing), and residential facilities for the elderly. (DIR, 1997).

**Construction workplaces** - Construction workers may encounter heavy concentrations of plaster/wallboard dust, adhesive VOCs, lead dust from surface preparation for painting, and fungal particles from mold remediation or flood-damaged buildings. Several organizations have published detailed guidelines for the protection of mold remediation workers (U.S. EPA, 2003b). However, very few studies have been conducted to determine the actual effectiveness of the guidelines in preventing health problems among these workers. A few case reports document illnesses among renovation workers.

**Roofing work** - Application of roofing asphalt is known to cause indoor air quality problems when contaminated air enters a building, often through ventilation system intake vents (Lynch and Kipen, 1998). Health complaints often continue well beyond the time of exposure.

**Aircraft cabins** - The pesticide permethrin is routinely sprayed on some international airliner routes to eradicate insects inside cabins. Flight attendants have become ill due to the exposure to permethrin; there is also concern about passenger safety. Airlines are not currently required to tell passengers of pesticide use (DHS, 2003). Aircraft exposures to biological contaminants and ozone also have been a concern (CITATIONS).

### Semiconductor industry

Although exposures experienced by workers in the semiconductor industry are essentially industrial exposures (as opposed to non-industrial), they are included here to illustrate concerns regarding exposures in non-traditional industrial environments, and the suspected health effects of some glycol ether compounds. These same glycol ethers have been detected in some consumer products and residential environments. Ethylene-based glycol ethers used in this field have been related to reproductive problems in animal species (Eskenazi *et al.*, 1995). Eskenazi *et al.* monitored reproductive activities and pregnancy rates of female workers in the semiconductor industry. The authors concluded that women employed in certain areas (fabs, dopant, and thin-film processes) have fewer pregnancies than other workers. Women exposed to ethylene glycol ethers had somewhat lower fecundability (probability of conception) than



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women unexposed to ethylene glycol ethers. Glycol ether levels were not recorded. In a similar study, glycol ethers and fluoride compounds used by workers in fabrication-rooms and photoresist processes accounted for increased risk of spontaneous abortions (Shanna *et al.*, 1995). Hammond *et al.* (1996) measured time-weighted average personal exposures for semiconductor workers. Workers that poured solvent or cleaned a coater cup experienced a higher exposure with a geometric mean concentration of 81 ppb of 2-ethoxyethanol. Workers who loaded and unloaded cassettes experienced a lower exposure with a geometric mean of 14 ppb 2-ethoxyethanol.

In summary, workplaces can pose a risk not experienced in residences or offices due to the nature of the activity that occurs there, or the type of sources present. While industrial exposures are moderated through the use of engineering controls, administrative controls, and personal protection gear when needed, indoor air quality problems in non-industrial workplaces have been less recognized and less studied. Nonetheless, some exposures in non-industrial workplaces have been shown to exceed guidelines for protecting the health of the general population, although levels of pollutants in those environments generally do not exceed occupational standard levels. Indoor air quality problems in non-industrial workplaces are not unique to California: in the past 25 years, the percentage of health evaluations that NIOSH (at the Centers for Disease Control and Prevention) has conducted related to indoor-air quality has increased from 0.5 percent of all evaluations in 1978, to 52 percent of all evaluations since 1990. This means that in those years, the evaluations related to air quality concerns have increased from one of every 200 evaluations to one of every two evaluations (Office of the Surgeon General, 1-13-05 news release).

### 3. COSTS OF INDOOR AIR POLLUTION

The health effects from indoor air pollution impose large costs on society, both economic and personal. The loss of human life, due to indoor air pollution constitutes a large economic and social cost. In addition, the medical costs of increased disease due to indoor air pollution, such as for cancer treatments, hospitalizations, chronic respiratory disease, and emergency room visits for asthma attacks and CO poisoning, are substantial. Student absenteeism, reduced worker productivity, and associated costs also result from indoor air pollution.

However, only very limited quantitative estimates on the costs of indoor pollution are available to date. Most of the available estimates are for a few specific indoor air pollutants that have been well studied, such as ETS and radon. There are many more indoor air pollutants that pose a significant health risk to the public, but the lack of cost information or other data for California precludes us from making even rough cost estimates here.

Estimates of the economic costs of some aspects of indoor air pollution have been made for the United States (Fisk, 2000; Mendell *et al.*, 2002). These studies allow order-of-magnitude estimates of the economic costs for California due to increased asthma symptoms and lost worker productivity in schools and office buildings. In addition, cost or value estimates can be derived for some of the health effects discussed in Chapter 2, in this report.

This information is used below, along with national and California-specific cost estimates for medical treatment and other costs (where available), to estimate the economic costs or value of specific health effects from indoor pollutants in California (U.S. EPA, 2002b; Thayer *et al.*, 2003). However, only the costs for selected indoor air pollutant exposures that have been reasonably well characterized in California are used for the final estimate. To account for inflation, cost estimates are adjusted to 2000 dollars using the relative increase in the urban consumer costs of the Consumer Price Index; for adjusting health care costs, the changes in medical care costs are used instead (USCIB, 2002). Table 3.1 presents the unit medical costs (direct and indirect) and economic valuations of premature deaths used in the following analyses.

#### 3.1 PREMATURE DEATH

Exposure to some air pollutants, primarily carbon monoxide, ETS, other carcinogens, and PM, can lead to near-term death or significantly increase the risk of premature death. The economic impact of premature death, or the value of a human life, has been estimated in the scientific literature and in environmental regulatory settings. The estimation methods include those based on: 1) an individual's willingness to pay to avoid a health risk, 2) the additional compensation demanded in the labor market for riskier jobs, and 3) society's willingness to pay to avoid a health or safety risk.

U.S. EPA (2002b) reviewed 26 value-of-life studies that used either the first or second method above, and confirmed the finding of a previous literature review that "most of the reasonable estimates of the value of life are clustered in the \$3.7 to \$8.6 million range."

**Table 3.1. Unit Costs for Health Effects.**

Outcome	Method <sup>1</sup>	Unit Cost in Year 2000 Dollars <sup>2</sup>
Premature death	WTP	\$6,330,000 <sup>3</sup>
Cancer medical costs (various types, survivor only)	COI	\$94,600
Lung cancer medical costs (weighted average for all cases)	COI	\$64,900
Asthma patient, direct medical and indirect costs	COI	\$640 <sup>4</sup>
Cardiovascular hospitalization	COI	\$15,200 <sup>6</sup>
Low birth weight, lifetime	COI	\$118,000
Otitis media (middle ear infection)	COI	\$360 <sup>5</sup>
Asthma, emergency room visit	COI	\$310 <sup>7</sup>
Asthma, chronic	WTP	\$33,000 <sup>7</sup>
Bronchitis, acute	WTP	\$59 <sup>7</sup>
Respiratory hospitalization (U.S.)	COI	\$11,000 <sup>6,7</sup>
Acute respiratory hospitalization (CA) Age < 18 Age ≥ 18	COI	\$11,800 <sup>6,7</sup> \$23,500 <sup>6,7</sup>

1. WTP: willingness to pay method; obtained from surveys. COI: cost of illness method; based from medical cost data and estimates of indirect costs such as lost workdays.
2. Unless noted otherwise, all values are averages from *Cost of Illness Handbook* (U.S. EPA, 2002b), after adjustment to 2000 dollars using the Consumer Price Index for medical care cost.
3. Midpoint value, adjusted for inflation using urban consumer costs.
4. From Weiss and Sullivan (2001), including direct medical and indirect costs. The 1998 U.S. estimates have not been adjusted to 2000 dollars.
5. From Gates (1996). The estimated annual U.S. cost of \$5 billion per year (in 1993 dollars) was divided by the estimated caseload of 14 million children under 5 years of age.
6. From a study of California costs by Thayer *et al.* (2003). Cost adjusted for inflation using the Consumer Price Index for medical care costs.
7. These values were not used in this analysis due to inability to separate hospitalization cases from doctor's visits in the literature.

Based on its review and input from the scientific community, U.S. EPA selected \$6 million (1999 dollars) as a point estimate for the "value of a statistical life," along with caveats for applying the study results to the general population. U.S. EPA (2000c) has used this point estimate in assessing the impact of regulations for diesel fuel and other pollutant sources. This point estimate is equivalent to \$6.33 million in 2000 dollars, and is used in the following sections to estimate the economic valuation of excess premature deaths due to indoor air pollution. The estimated valuation of premature death from indoor air pollution in California is discussed below and summarized in Table 3.2.

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Another approach to estimating the economic cost from deaths due to indoor air pollution is to base the estimate on court decisions and insurance awards, such as those from CO poisoning. However, nearly all cases of fatal and non-fatal CO poisoning are settled out of court, and utility companies are not required to track such cases; thus, there is little reliable data available on these legal settlements.

### 3.1.1 Deaths From CO Poisonings

Based on the DHS review of coroners' reports in the 1980's, an average of 30-40 Californians die each year from accidental CO poisoning (Girman *et al.*, 1998; Liu *et al.*, 1993a, 2000). On average, about 2/3 of those deaths (about 20-26, or 23 average) documented in the DHS study were due to appliance-related causes such as faulty furnaces, gas ovens used for space heating, and charcoal grills used indoors. This estimate has a high degree of certainty relative to other pollutant risk estimates because the CO death estimates are based on coroners' reports that clearly identify CO poisoning deaths. The economic value of these lost lives in California is estimated to be \$130-160 million per year (2000 dollars), or about \$150 million on average, as shown in Table 3.2.

Although the rate of CO deaths in the U.S. population has declined over the years (CPSC, 1997), the actual numbers of CO poisoning cases in California may have increased since 1990. Based on California's population growth alone – from 30.38 million in 1990 (USCB, 1990) to 33.87 million in 2000 (DOF, 2002ab), a 14% increase – one might expect the number of deaths to have increased by a similar percentage. In addition, much of this population growth has occurred in the inland and foothill regions of California, which have longer, colder winters than coastal areas, and thus, would have a greater likelihood of CO poisoning due to more frequent use of combustion appliances for space heating.

However, trends toward increased use of CO alarms and testing of combustion appliances could potentially have reduced the number of appliance-related CO poisonings in California since 1990. CO poisonings at the national level have declined (CPSC, 1997), which may be due to lower pollution emissions from motor vehicles and the increased heavy marketing of inexpensive CO alarms for use in homes, motor homes, and boats. The state's program for weatherizing existing, low-income homes has required installation of CO alarms and combustion appliance safety testing, as discussed in Section 4.3.2.

Overall, based on the factors in both directions discussed above, the current number of fatal CO poisonings in California due to appliance-related causes is assumed similar to that in 1990. It is likely that the risk of CO poisoning remains higher than average in older homes with older gas appliances, in homes with propane appliances, and in some socioeconomic groups that use unvented gas stoves or charcoal grills indoors more frequently.

**Table 3.2. Estimated Valuation of Mortality  
Attributed to Indoor Air Pollution in California**

End Point	Excess Premature Deaths (cases/yr) <sup>1</sup>			Valuation per case <sup>2</sup> (\$ Million)	Cost per Year <sup>3</sup> (\$ Billion)		
	Low	Average	High		Low	Average	High
CO: Poisoning <sup>4</sup>	20	23	26	6.33	0.13	0.15	0.16
VOCs: Lung cancer <sup>5</sup>	115	115	115	6.33	0.73	0.73	0.73
ETS: Lung cancer <sup>6,7,8</sup>	380	380	1,020	6.33	2.4	2.4	6.5
ETS: Heart disease <sup>7,8</sup>	1,700	3,600	5,500	6.33	11	23	35
Radon: Lung cancer <sup>9</sup>	600	1,500	3,500	6.33	3.8	9.5	22
Mold and Moisture: Asthma and allergy <sup>10</sup>	NA	NA	NA	NA	0.031	0.031	0.031
<b>TOTAL<sup>11</sup></b>					<b>18</b>	<b>36</b>	<b>64</b>

1. Low and high estimates are based on a range or confidence intervals, where available. Average estimates are based on mean values from available estimates or ranges.
2. From *Cost of Illness Handbook* (USEPA, 2002b), adjusted to 2000 dollars.
3. Costs per year are rounded to 2 significant figures.
4. Case estimate from coroner's data in California (Liu *et al.*, 2000).
5. Case estimate from average values in California Comparative Risk Project (CCRP, 1994), updated. See Section 2 and Appendix II of this report.
6. From best risk estimate rather than average risk estimate; for spousal smoking only (OEHHA, 2004). Adjusted to 2000 population total. This risk estimate is probably an overestimate because it assumes ETS exposure and death rates in California are the same as those for the U.S.
7. From OEHHA (2004).
8. OEHHA (2004) is currently incorporating comments from the independent Scientific Review Panel on this report.
9. From Waldman (2004), based on USEPA (2003c), with adjustments for smaller population, lower average residential radon concentrations, and lower smoking prevalence rates in California.
10. Cost estimates taken from national cost estimated by Weiss and Sullivan (2001) of \$2 billion (in year 2000 dollars), scaled to 12% for California estimate, and multiplied by fraction of asthma risk attributed to mold and moisture in three large epidemiology studies (0.13).
11. These totals do not include death from exposures to other types of indoor PM, which could be substantial but are not currently quantifiable due to lack of appropriate studies.

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### 3.1.2 Deaths From Volatile Organic Compounds (VOC) Exposure

As discussed in Section 2 and Appendix II, about 230 excess cancer cases due to VOCs from indoor exposures are estimated to occur in California each year. This estimate is based on the results of the California Comparative Risk Project (CCRP, 1994) that have been updated to reflect reductions in formaldehyde exposures and increased population growth as of the year 2000. These are conservative estimates relative to the total cancer burden from indoor carcinogens, because they do not include the additional, significant cancer risk from radon, and the risk from many other carcinogens also found in indoor air and house dust, such as acetaldehyde, PAHs other than B(a)P, phthalates other than DEHP, and asbestos.

Half of the VOC-related cancer cases attributed to indoor air (115) per year are estimated to result in premature death. U.S. EPA (2002b) assumed this same fraction of fatal cases within five years of diagnosis when estimating the costs of typical cancer treatment. Actual premature death rates could be higher than one-half of the cases because cancers caused by these air pollutants typically result in lung, organ, or blood cancers, which are more difficult to detect and treat, and thus, are more deadly. The estimated value of premature deaths from the 115 excess cancer deaths in California totals \$730 million per year, as shown in Table 3.2. This estimate does not explicitly include the much smaller costs of cancer treatment, which are discussed below.

### 3.1.3 Deaths from Environmental Tobacco Smoke Exposure

ARB/OEHHA (2004-2005) estimate that exposure to ETS from spousal smoking in California in 2003 produced 400 premature deaths (range 411-1,064) due to lung cancer. These estimates are based on the best risk estimate rather than the average risk estimate. They also are based on smoking prevalence rates in the U.S., and scaled down to reflect that California contained 12% of the U.S. population.

To adjust the ARB/OEHHA estimate back to the year 2000 population, the lung cancer risk estimate was multiplied by 0.96. This yields an estimated number of 380 excess lung cancer deaths cases per year from ETS exposures. The estimated value of premature deaths from the 380 excess lung cancer deaths (range 390-1,020) in California totals \$2.4 billion per year, as shown in Table 3.2. This estimate does not explicitly include the much smaller costs of cancer treatment, which are discussed below

For ETS effects on ischemic heart disease, ARB/OEHHA (2004-2005) estimated that ETS exposure resulted in 1,700-5,500 (average = 3,600) premature deaths from heart disease. This estimate was based on 1999 ETS exposure data and 2000 mortality data for California. The estimated value of 3,600 deaths is \$23 billion, as shown in Table 3.2.

The estimated numbers of premature deaths from ETS exposure, and the morbidity effects discussed below, probably overestimate current risk levels for two reasons. First, ETS exposure is likely much lower in California compared to the U.S. The CDC (2004) found adult and adolescent smoking prevalence in California was about 16% in 2001, while the U.S. prevalence rate was 22%. In addition, cigarette consumption by California adults was found to be about half of the U.S. average in 2001-2002 (DHS, 2002a). Secondly, the estimates include home and workplace exposure, but California's workplace smoking ban has virtually eliminated ETS exposure in enclosed workplaces, with a few exceptions. By 1999 over 93% of indoor workers reported having a smoke-free workplace (DHS, 2002b). On the other hand, newer data on the health effects of ETS may increase the unit risk of ETS for death and disease. For example,

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ARB/OEHHA (2004) estimate that ETS increases the risk of breast cancer by about 30%. ARB/OEHHA currently revising the report to reflect the comments of the external Scientific Review Panel on breast cancer and other topics, and those revised estimates may be included in this report at a later date.

These costs of ETS-related health effects could be prevented if people smoked only outdoors away from other people, or stopped smoking. However, to achieve these potential cost savings, further efforts are needed to change peoples' smoking behaviors, and to improve awareness of and control of other indoor and outdoor asthma triggers in California (Meng *et al.*, 2003).

### 3.1.4 Deaths from Radon Exposure in Homes

The DHS has estimated that 1,500 excess lung cancer deaths per year are attributed to the residential radon exposure in California (Waldman, 2004), as discussed in chapter 2. This first-order estimate is based on the U.S. EPA's (2003c) national estimate of 21,000 excess lung cancer deaths (90% CI: 9,000 to 50,000). To calculate the residential radon risk for excess lung cancer in California, the national estimates were multiplied by the fraction of the U.S. population in California (34 million out of 281 million, or 12%; U.S. Census 2000 data). The national estimates were also multiplied by the ratio of average radon concentrations (CA: 1 pCi/l; U.S.: 1.25 pCi/l; ratio of 0.8) and the smoking prevalence in California (CA: 16.4%; U.S.: 22.5%, CDC, 2004). This yielded an estimate for the radon-induced lung cancer death in California of about 1,500 (90% CI: 600-3,500) deaths per year. This may overestimate the excess deaths because recent measurements in the Sierra Nevada region indicate that this region has much lower radon levels than that reported from the previous statewide survey (3 vs. 25%), and that the statewide average may be lower than previously estimated (Tsai and Waldman, 2005). The estimated value of premature deaths from the 1,500 excess lung cancer deaths in California attributable to radon totals \$9.5 billion per year, as shown in Table 3.2.

### 3.1.5 Deaths from Mold and Moisture-related Problems in Homes

Weiss and Sullivan (2001) estimated that the costs of excess death in children and adults due to asthma in the U.S. was \$2 billion, in year 2000 dollars. Scaling this to reflect that 12% of the U.S. population resides in California gives an estimated cost for asthma deaths in California of \$0.24 billion. As discussed below in the Medical Costs section, the fraction of these health effects attributed to mold and moisture problems in homes is 0.13. This fraction is an average of four large, well-conducted epidemiology studies of adults and/or children (shown in Table 3.4). Multiplying the estimated cost of asthma in California by this fraction yields an estimated cost of \$31 million attributable to excess death due to residential mold and moisture problems. This estimate does not include other indoor allergen sources such as dust mites, pets, cockroaches, and chemical emissions.

## 3.2 MEDICAL COSTS

Illness and disease caused by indoor air pollution include the production of new asthma cases (induction), exacerbation of asthma symptoms, development of other respiratory disease and symptoms, and induction or exacerbation of allergies. These impacts of indoor air pollution also affect the quality of a person's life in terms of reduced or limited activities, limited employment opportunities, and reduced productivity.

The cost of illness and disease has been estimated in various ways. These include methods based on medical costs, work-related costs, education-related costs, and willingness of

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individuals to pay to avoid the anxiety, pain, suffering, and other health risks (U.S. EPA, 2002b). The following section uses medical costs shown in Table 3.1 to estimate the medical costs of indoor air pollution in California, but generally excludes the indirect costs of reduced productivity of individuals and employees. The estimated medical costs are summarized in Table 3.3.

### 3.2.1 CO-related Hospitalization

About 175-700 emergency room visits and hospitalizations (midpoint of 438) due to non-fatal CO poisoning are estimated to occur in California each year, on average (see Section 2.2.4.2). Emergency room visits cost about \$442 per visit, based on costs for asthma room visits to emergency rooms and adjustment to 2000 dollars (U.S. EPA, 2002b). Therefore, the annual cost of emergency room visits due to CO poisoning in California ranges from \$77,000-310,000, or an average of about \$190,000, as shown in Table 3.3.

The estimated number of emergency room visits due to CO is conservative because CO poisoning can be misdiagnosed as a viral flu illness or general fatigue. The estimated cost for each case of non-fatal CO poisoning is conservative because it does not include several types of costs that could be substantial, such as the costs for lost school and work days, hyperbaric oxygen treatment for severe poisoning, long-term neurological and developmental damage to many victims of sub-lethal CO poisoning, and the estimated hundreds to thousands more CO poisoning cases per year that do not result in hospitalization.

### 3.2.2 VOC-related Cancer Treatment

The number of excess cancer cases from VOC exposures was estimated above to be 230 cases per year, and one-half of those cases (115) were assumed to survive, as discussed above. The average cost of medical treatment in the U.S. for the 13 most common types of cancer, when assuming a 50% death rate at five years and no discounting, is \$82,581 in 1996 dollars (U.S. EPA, 2002b) or \$94,619 in 2000 dollars. This treatment cost estimate is conservative because it does not consider recent changes in cancer treatment that can be very expensive, such as the bone marrow transplant procedure and new pharmaceutical treatments. At this treatment cost per cancer case, the total cost for medical treatment for 115 cases from VOC exposure was estimated to be \$11 million, as shown in Table 3.3.

### 3.2.3 ETS-related Illness and Hospitalization

As discussed above, ETS exposure was estimated to result in 380 excess deaths per year from lung cancer. To estimate the medical costs of this impact, the approach used above for VOCs was used, except that the average cost of medical treatment for lung cancer alone was used instead: \$56,624 in 1996 dollars (U.S. EPA, 2002b) or \$64,900 in 2000 dollars. Costs for lung cancer only were used because ETS exposure is primarily associated causally with lung cancer, while VOCs are causally associated with several other types of cancer. This cost is based on the total costs for non-survivors and survivors over 10 years, weighted annually for survival rates and discounted at 5%. The total cost of cancer treatment for the 380 cancer cases from ETS exposures was estimated to be \$25 million (in 2000 dollars), as shown in Table 3.3.

For estimating costs due to hospitalization for heart disease due to ETS exposure, an approach similar to that used above for VOCs was used. However, in this case, the average cost of medical treatment for heart disease (\$15,200) from Table 3.1 was used. The total cost of



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hospitalization for 3,600 heart disease cases attributed to ETS exposures was estimated to be \$55 million (in 2000 dollars), as shown in Table 3.3.

ETS exposure in California was estimated to have resulted in at least one asthma episode in 31,000 (range of 24,000-40,000) non-smoking children under 18 years of age over the last 12 months (ARB/OEHHA, 2004-2005). This estimate was based on a meta-analysis of 29 epidemiological studies, and an attributable fraction based on California data for population and ETS exposure prevalence for 2000. The estimate includes exacerbation of asthma in both new and existing asthma patients in the last 12 months. These health effects were primarily observed in infants and older children, but they may also increase the risk of health effects later in life. The average unit cost for each asthma patient in the U.S. was estimated to be \$640 per year, based on national data for 1998 (Weiss and Sullivan, 2001). This unit cost includes direct medical and indirect costs for all age groups, and is about twice the unit cost for asthma treatment in an emergency room (\$310). Actual costs for asthma episodes are likely to be higher because the medical costs of asthma treatment are rising rapidly and the costs of upper and lower respiratory conditions that occur along with asthma are not included (Weiss and Sullivan, 2001). Multiplying the number of asthma episodes by the unit cost for asthma patients (\$640) from Table 3.1, yields an estimated cost of \$19 million per year (1998 dollars) in direct medical costs and indirect costs, as shown in Table 3.3.

ARB/OEHHA (2004-2005) estimate that ETS exposure resulted in 1,600 cases of low birth weight. Again, the case numbers multiplied by the unit medical cost shown (\$118,000) in Table 3.1. The estimated cost of the 1,600 cases of low birth weight infants was \$190 million. This unit medical cost is likely to be an underestimate of actual costs because credible data are lacking for the first two years of life when intensive medical care and additional hospitalization are most likely, and for some age groups where costs were extrapolated from others (U.S. EPA, 2002b). In addition, cost data for non-medical expenses over a lifetime were lacking; these costs could be substantial as well.

ARB/OEHHA (2004-2005) estimated that 4,700 cases of premature delivery result from ETS exposure in California each year. Premature delivery is a major cause of infant mortality and infant medical expenses in the U.S., especially among African-Americans (MOD, 2003). Because the large majority of premature infants have low birth weights (Mattison *et al.*, 2001), most of the medical costs of premature delivery are included in the cost estimate above for low birth weight due to ETS exposure. However, the increased costs of premature death, long-term disability, and indirect expenses due to premature delivery are not available. OEHHHA is currently revising the report to reflect the comments of the external Scientific Review Panel on developmental effects and other topics, and those revised estimates will be included in this report at a later date.

ARB/OEHHA (2004-2005) also estimated that 51,700 cases of otitis media (middle ear infection) in children were associated with ETS exposure in California. This estimate is based on ETS exposure of California children in 1999, and 2000 California population data. Gates (1996) estimated that the direct and indirect cost of otitis media totaled \$5 billion dollars per year in 1993. Dividing this cost by the estimated caseload of 14 million children under the age of 5, the primary patients for this diagnosis, yields a unit case cost of \$360 per year (1993 dollars). This unit cost is an underestimate because it does not include all medical costs, the potential cost of impacts on children's learning and development, and the increase in medical costs and consumer costs since 1993. This unit cost is relatively minor compared to, and somewhat redundant with, the costs estimated above for new cases of chronic asthma (\$33,000); it is provided here to address the impacts on persons with pre-existing asthma. Multiplying the

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estimated number of cases by the unit cost (\$360) yields an estimated cost of \$19 million per year (1993 dollars), as shown in Table 3.3.

**Table 3.3. Estimated Annual Medical Costs of Indoor Air Pollution in California.**

Indoor Pollutant and Health End Point	Average Cases per Year <sup>1</sup> (Range)	Medical Cost per Case <sup>1,2</sup> (\$)	Average Cost per Year <sup>3</sup> (\$ millions)
CO: poisoning	438 (175-700)	442	0.19
VOCs: cancer	115	94,600	11
ETS: lung cancer <sup>4,5</sup>	380	64,900	25
ETS: heart disease <sup>4,5</sup>	3,600 (1,700-5,500)	15,200	55
ETS: asthma episodes <sup>4,6</sup>	31,000 (24,000-40,000)	640	20
ETS: low birth weight <sup>4</sup>	1,600	118,000	190
ETS: otitis media visits <sup>4</sup>	51,700	360	19
Radon: lung cancer <sup>7</sup>	1,500	64,900	97
Mold & moisture: asthma and allergy <sup>8</sup>	NA <sup>9</sup>	NA	190
<b>Total<sup>10</sup></b>			<b>610</b>

1. Original data were adjusted to 2000 dollars and 2000 population where necessary.
2. Medical cost values were taken from Table 3.1 and do not include indirect costs, unless noted otherwise. Thus, cancer treatment cost does not include costs for outpatient prescription medications and nursing home care below the skilled level.
3. Rounded to two significant figures.
4. Case estimate from OEHHA (2004).
5. Medical costs for fatal cases of cancer or heart disease also are implicitly included in death valuations, but are generally an insignificant amount relative to the total valuations.
6. The case estimate is for children under 18 years of age with at least one asthma episode in the past 12 months.
7. From Waldman (2004), based on U.S. EPA (2003c) with adjustments for smaller population, lower average residential radon concentrations, and lower smoking prevalence rates in California.
8. Cost estimates taken from national estimates for medical and indirect costs by Weiss and Sullivan (2001), adjusted to 2000 dollars, scaled to 12% for California estimate, and multiplied by fraction of asthma risk attributed to mold and moisture in four large epidemiology studies (0.13). Includes estimate of \$60 million for indirect costs, such as lost workdays, lost school days, and reduced housekeeping.
9. NA = not available.
10. These totals do not include disease from exposures to other indoor PM, which could be substantial but are not currently quantifiable.

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### 3.2.4 Radon-Related Cancer Treatment

As discussed above, residential exposure to radon was estimated to result in 1,500 excess deaths per year from lung cancer. To estimate the medical costs of this impact, the approach used above for ETS was used; \$64,900 in 2000 dollars was used for the average cost of medical treatment for lung cancer (U.S. EPA, 2002b). Only costs for lung cancer were used because radon exposure is primarily associated causally with lung cancer. The total cost of cancer treatment for the 1,500 cancer cases attributed to indoor radon exposures was estimated to be \$97 million (in 2000 dollars), as shown in Table 3.3.

### 3.2.5 Medical and Indirect Costs Associated with Indoor Mold and Moisture-related Problems in Homes

Available data demonstrate a relatively strong and consistent association of dampness and mold in buildings with an increase in lower respiratory symptoms that are often considered evidence of asthma exacerbation (IOM, 2004). The specific biological, chemical, or physical agent responsible for the health-relevant exposures from indoor dampness and mold are not fully understood. Few large studies of mold, allergens, and other biological contaminants have been conducted in office buildings, schools, and other non-residential buildings, so this section focuses on residential exposures.

To estimate the cost of asthma attributable to dampness or mold, the fractions of asthma attributable to dampness or mold in apartments and homes in the U.S. and California are multiplied by estimates of the cost of asthma. Four large studies of the health risks of building dampness and mold for adults and/or children have been conducted in the U.S. and Canada, as shown in Table 3.4. Each of these studies controlled for the effects of many factors other than dampness and mold. Most of the study areas have moderate or cold climates similar to California, and the three studies from Canada or the U.S. had very similar odds ratios for dampness or mold. In addition, southern California homes in the ARB's Children Health Study (Taylor, 2004) were found to have reported dampness or musty odors at rates similar to those reported in the U.S. and Canadian studies (Spengler *et al.*, 1994; Dales *et al.*, 1991ab) – over 30%. Thus, the results of the studies shown in Table 3.4 can be used to make estimates for California.

Equation 1 was used to estimate the fraction of asthma attributable (AF) to dampness and mold, using odds ratios (OR) and prevalence rates (p) specific to each study population:

$$AF = p(OR-1) / (p(OR-1)+1) \quad [Equation 1; Lilienfeld and Lilienfeld, 1980]$$

Table 3.4 shows the calculations of attributable fraction<sup>1</sup> using odds ratios, rather than relative risks, from the results of these four major studies. The average attributable fraction of asthma due to dampness or mold in homes is estimated to be 0.13.

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<sup>1</sup> The attributable fraction is normally calculated using the relative risk value, but odds ratios can be used instead where the outcome prevalences are relatively low. For example, with prevalence rates of 11.4% for asthma symptoms and 16.9% for lower respiratory symptoms in the study by Spengler *et al.* (1994), the relative risks are approximately equal to the odds ratios. Thus, the attributable fraction can be estimated using odds ratios (ORs) in place of relative risks (RRs).

**Table 3.4. Fraction of Asthma Attributable to Mold or Dampness in Houses**

Study and Condition	Study Population	Prevalence of Condition (%)	Asthma Symptoms	
			Adjusted Odds Ratio (95% CI)	Estimated Attributable Fraction
Spengler <i>et al.</i> (1994), mold or mildew	12,842 U.S. children ages 9-11	36	1.39 (1.23-1.57)	0.12
Dales <i>et al.</i> (1991a), dampness or mold	13,495 Canadian children ages 5-8	38	1.45 (1.23-1.71)	0.15
Dales <i>et al.</i> (1991b), dampness or mold	14,700 Canadian adults	38	1.56 (1.25 -1.95)	0.18
Zock <i>et al.</i> (2002), mold or mildew last year	19,218 adults, 38 centers in U.S., Europe, Australia, India, New Zealand	22	1.28 (1.13-1.46)	0.06
<b>AVERAGE</b>				<b>0.13</b>

To estimate the national cost of asthma, two estimates of asthma costs (Weiss and Sullivan, 2001; Smith *et al.*, 1997) were reviewed. The estimates of direct costs from the two studies are similar. However, the estimate of medical and indirect costs based on Weiss and Sullivan (2001) are more recent than those from Smith *et al.* (1997), and they do not exclude persons in the military, schools, and other institutions. Thus, the Weiss and Sullivan results may better reflect the current costs of treatment and medications, and are used here.

The Weiss and Sullivan estimates were adjusted to 2000 dollars and 2000 population using the data on population growth and medical care and general inflation. No adjustment was made for changes in asthma prevalence because it is not clear that asthma prevalence has changed since 1994 (Mannino *et al.*, 2002). It was also assumed that the prevalence of asthma in California is similar to the average prevalence of asthma in the U.S. Data in IOM (2004) indicate that self-reported asthma prevalence in California was 7.1% versus 6.4% for the U.S.; however, more recent data (Rhodes *et al.*, 2002) indicate that the prevalence of asthma in 2002 was 6.4% in California versus 7.5% for the full U.S. Thus, it is not clear that asthma prevalence in California differs from that for the U.S. at this time.

The updated costs for the full U.S. were then multiplied by the 0.12, the percentage of U.S. population that resided in California in 2000. As shown in Table 3.5, the estimated premature death costs are \$240 million, as discussed in the premature death section above. The estimated medical costs and indirect costs (such as lost work days and school days) of asthma in California are \$980 million and \$460 million, respectively.

To estimate the asthma costs attributed to indoor mold and moisture problems, these costs of asthma in California were multiplied by 0.13, the attributable fraction of asthma from mold and dampness that was calculated above. This yields the estimated medical and indirect costs of \$130 million and \$60 million, respectively, for a total of \$190 million, shown in Table 3.3. This estimate does not include the costs of other indoor allergen sources such as dust mites, pets, cockroaches, and chemical emissions.

**Table 3.5. Updated Estimates of Annual Cost of Asthma.**

Population Group	Cost in U.S. (\$ Billions) <sup>1</sup>				Cost in California (\$ Billions) <sup>1</sup>			
	Death	Medical	Indirect <sup>2</sup>	Total	Death	Medical	Indirect <sup>2</sup>	Total
Children	--	--	1.2	--	--	--	0.14	--
Adults	--	--	2.6	--	--	--	0.31	--
Both	2.0	8.2	3.8	13.9	0.24	0.98	0.46	1.7

1. In 2000 dollars. Row and column totals may not add precisely due to rounding of numbers. Source of U.S. estimates: Weiss and Sullivan (2001). California costs were estimated to be 12% of U.S. costs, based on the relative population sizes.
2. Indirect cost elements include loss of work, loss of school, and reduced housekeeping.

Extensive data on are not available on the costs of asthma that are preventable, but a large portion would appear to be readily preventable. The particle sources considered in this analysis are tobacco smoking, pets, use of gas stoves for heat, dust mites, cockroaches, and dampness/mold. Behavioral changes can eliminate all indoor tobacco smoking, indoor pets in homes of people with pet allergies, and use of gas stoves for heat in the homes of asthmatics. Dust mite allergen levels can be diminished by reducing indoor humidity and by surface cleaning; however, studies of dust mite remediation measures have had only moderate success. Cockroach infestations can be reduced substantially using pest management methods. Mold contamination in buildings can be reduced by preventing and remediating dampness problems.

Many dampness problems, probably a majority of serious problems, result from water leaks that could be prevented through better building maintenance and improved design and construction. These measures would also reduce the costs of dampness-caused mold contamination and degradation of building materials. Better ventilation and use of dehumidifiers could reduce dampness problems that result from high indoor humidity. Thus, with proper measures, it is probably feasible to eliminate at least 50% of the particle exposures that contribute to asthma exacerbation, and likely more.

### **3.3 PRODUCTIVITY COSTS OF INDOOR AIR POLLUTION**

Sick building syndrome is a collection of non-specific symptoms such as eye, nose, skin, and throat irritation; headache; fatigue; and skin rash that have no known cause. Inadequate building ventilation, as indicated by elevated indoor CO<sub>2</sub> concentrations, as well as elevated indoor levels of VOCs, elevated levels of biological contaminants, and other environmental stressors have been implicated as potential causes of sick building syndrome.

Fisk (2000) estimated the economic impacts of sick building syndrome in the U.S. due to reductions in worker productivity. Several field and laboratory studies of office buildings and school buildings were reviewed; the performance reduction for specific tasks ranged from 3-5%. The midpoint value of 4% was reduced to 2% to estimate the overall productivity reduction throughout the day. Using this conservative estimate of a 2% preventable reduction in worker productivity due to sick building syndrome, the estimated cost savings for the U.S. in 1996

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dollars were \$10-30 billion (\$11-33 billion in 2000 dollars). An independent group of scientists who reviewed the literature and assessed the impacts of indoor air quality on worker health and productivity for the National Institute of Occupational Safety and Health estimated the same costs (Mendell *et al.*, 2002).

To estimate the costs of sick building syndrome in California, this \$11-33 billion U.S. estimate by Fisk can be scaled to reflect California's portion of the U.S. population, or 12%. This would yield a California estimate of \$1.3-4.0 billion per year, for an average of \$2.6 billion per year.

Kats (2003) used the 2% preventable reduction in worker productivity estimate by Fisk (2000) to estimate an avoidable cost of sick building syndrome in California. Using California-specific data where available, Kats estimated this avoidable cost to be \$9 billion per year (\$6 billion in wages and \$3 billion in worker benefits). Using only a 1% lost productivity value, Kats calculated that sick building syndrome reductions in State of California buildings were equivalent to a present value of \$37-\$55 per square foot of building space over 20 years, compared to average energy costs of \$1.47 per square foot for state buildings. The avoidable cost estimate of \$9 billion for California is much larger than the estimate derived from Fisk because it includes employee benefits to reflect the workers' full market value, and it reflects the higher percentage of office workers and the higher salaries in California compared to the U.S.

For estimating the costs of sick building syndrome in California's school and office buildings, we use the more comprehensive and California-specific approach of Kats. We modified his estimate by using more recent data and conservative estimates of worker benefits costs, as follows. California has 7 million workers in offices or schools, and their average salary is \$43,000 per year in 1998 dollars (Kats, 2003). Sick building syndrome symptoms are again conservatively estimated to cause a 2% decrease in worker productivity. Multiplying these values together yields a cost of about \$6 billion per year. For U.S. white collar workers in 1999, salary accounted for 72.8% of their total compensation on average, while benefits such as health insurance and retirement accounted for 27.2% (USCB, 2002). Dividing this benefit percentage by the salary percentage yields a ratio of 0.37 as the additional fraction for compensation as benefits. Multiplying 0.37 by the salary estimate of \$6 billion yields an estimated overhead cost of \$2.2 billion. This is a conservative estimate because it does not include other overhead costs to the employer such as training, equipment, and travel. Combining these salary and benefit cost estimates yields a total cost estimate of \$8.2 billion, or \$8.5 billion in 2000 dollars, due to the avoidable impacts of sick building syndrome on worker productivity in California.

### 3.4 SUMMARY OF ESTIMATED COSTS

The combined cost of both fatal and non-fatal impacts due to indoor air pollution in California homes, schools, and non-industrial workplaces is substantial; it is estimated at **\$45 billion per year**, as shown in Table 3.6 below. The annual valuation of premature death attributable to indoor air pollution is estimated to total about \$36 billion. The costs attributed to ETS and radon dominate the total cost. However, this is not because they are necessarily the predominant health hazards from indoor air in California, but because those are the two pollutants out of many indoor air pollutants for which there is sufficient data to estimate risk and costs. The cost estimates in this report do not include other indoor air pollutants that can increase the risk of premature death, and any synergistic effects among indoor and outdoor pollutants. Examples of these other pollutants include: PM from wood smoke, other toxic substances emitted from materials and products, and biological pollutants such as mold, bacteria, pollen, and animal

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allergens. Therefore, the actual total valuation of death and disease costs are likely to be even higher than estimated here.

The quantifiable medical costs (direct and some indirect) due to some indoor air pollutants total more than \$0.6 billion per year, with a large portion of the costs attributable to mold and other moisture-related allergens and sum of all ETS-related medical costs. Again, this is certainly a very low estimate: the cost estimates for disease do not include of the potential losses due to other indoor allergens, CO poisoning's long-term effects, reduced student performance, lost earnings opportunity, unpaid caregivers, and human suffering. Finally, the cost of reduced worker productivity due to indoor air pollution (sick building syndrome) that could be prevented is estimated to be \$8.5 billion per year. As discussed in the next section, case studies have documented that measures to reduce indoor air pollution in homes and schools can have immediate and cost-effective benefits on human health and student performance.

### 3.5 BENEFITS OF IMPROVING INDOOR AIR QUALITY

Several case studies and demonstration programs have documented the economic, productivity, and health benefits that can accrue by improving IAQ. The following examples and other available examples provide empirical data on the benefits achieved in different types of buildings.

#### Residential Buildings

The Seattle Healthy Home Program has found that home visits to low-income households by a trained community health worker can reduce asthma symptom significantly and cost-effectively (Takaro *et al.*, 2004; Krieger *et al.*, 2005). This program reduced medical costs for asthma treatment significantly, in both the low-intensity version (one visit over the year) and the high-intensity version (multiple visits over one year). The observed marginal cost savings were \$1,316 to \$1,849 per patient over four years. These savings do not reflect the reduced number of emergency room visits and the reduced risk of asthma-related deaths.

The "Healthy Neighborhoods Program" of New York State inspected over 45,000 homes for health and safety problems, and intervened where necessary (HUD, 1999; NYS, 1999). Interventions addressed lead-based paint hazards, fire safety, sources and detection of carbon monoxide, and asthma-related conditions. Interventions were generally simple and low cost, such as providing working smoke detectors and batteries, CO detectors, and furnace filters. The program results showed that the visits to each home cost \$132 per unit. However, the benefits for lead poisoning prevention, asthma reduction, and burn prevention alone were worth at least \$285 per unit, excluding estimated benefits associated with reduced injury, CO poisoning, and fire.

#### Schools

Office visits for asthma inhaler use dropped by 50% at two elementary schools in San Francisco Unified School District (USD) after the *IAQ Tools For Schools* program was pilot tested (U.S. EPA, 2000a). In addition, fewer asthma episodes occurred, and fewer students brought asthma medications or inhalers to school.

A study of Chicago and Washington, DC schools found that better school facilities can add three to four percentage points to a school's standardized test scores, even after controlling for demographic factors (Schneider, 2002). This and other studies reviewed by Kats (2003) and Fisk (2000) confirm a widely held, common sense perception that the physical quality of the classroom environment greatly affects how well children learn.

**Table 3.6. Summary of Estimated Costs of  
Some Indoor Air Pollution in California**

Health End Point	Health Valuation: Premature Death <sup>1,3</sup> (\$ Billions/yr)	Medical Cost <sup>2,4</sup> (\$ Billions/yr)	Lost Productivity Cost <sup>2,3</sup> (\$ Billions/yr)	Total Cost (\$ Billions/yr)
CO: poisoning	0.15	<0.001	NA	0.15
VOCs: cancer	0.73	0.011	NA	0.74
ETS: lung cancer	2.4	0.025	NA	2.4
ETS: heart disease	23	0.055	NA	23
ETS: asthma episodes	NA	0.020 <sup>5</sup>	NA	0.020
ETS: low birth weight	NA	0.19	NA	0.19
ETS: otitis media	NA	0.019 <sup>5</sup>	NA	0.019
Radon: lung cancer	9.5	0.097	NA	9.6
Mold and moisture: asthma and allergies	0.031	0.19 <sup>5</sup>	NA	0.22
Sick building syndrome	NA	NA	8.5	8.5
<b>TOTAL<sup>6</sup></b>	<b>36</b>	<b>0.6</b>	<b>8.5</b>	<b>45</b>

1. From Table 3.2.
2. Estimates are based on average or mid-point of incidence rates of mortality and morbidity from previous tables, and estimates of productivity discussed in the text. Values are rounded to two significant figures.
3. Original data were adjusted to year 2000 dollars and year 2000 population, except where noted otherwise in previous tables.
4. From Table 3.3.
5. Includes indirect costs such as lost work days, lost school days, and travel expenses.
6. Totals are rounded to 2 significant figures. These totals are likely low because conservative cost estimates were used, and quantitative information is not readily available for many known impacts of indoor air pollution, such as for indoor PM and many indirect costs of health effects.

An analysis of two school districts in Illinois, one small and one large, found that student attendance improved by 5% after incorporating cost effective indoor air quality improvements – regardless of school district size (Healthy Schools Campaign, 2003).

Clovis USD near Fresno, and Everett USD near Seattle, have used the *IAQ Tools for Schools* program and a rapid complaint response approach using a portable indoor air monitoring kit. Additional staff were not needed to implement the program. his program has quickly paid for



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equipment by reducing the number of complaints by up to 60%, and by reducing the cost for environmental consultants (Poytress, 2003; Jefferis, 2004). It has also greatly improved the credibility of the school maintenance program with school staff and parents of students.

Indoor air quality is one of several important indoor environmental factors affecting student performance. In a study of over 8,000 students in third through sixth grade in Fresno, Heschong (2003) investigated the effect of daylight and other indoor environmental quality effects on student test scores over an academic year. As expected, commonly recognized factors such as teacher characteristics, number of computers, or attendance rates were found to be significant in predicting student performance. However, physical characteristics such as indoor air quality, ventilation, acoustics, and especially daylighting were found to be equally significant, if not more significant.

The economic benefits of improved indoor air quality in schools, in terms of improved student productivity and health has not been estimated. The potential benefits of improved productivity, even assuming a slight increase in test scores and intelligence quotients (IQ), could be quite large.

### Office Buildings

Recent experiments in office buildings have shown that office worker performance could be significantly increased over the short term by removing common indoor sources of air pollution, such as floor coverings, used supply air filters, and personal computers (Wyon 2004). Alternatively, keeping these pollutant sources in place while increasing the ventilation rate from 1.5-3 liters per second (lps) to 10-30 lps per person (3-6 cfm/person to 20-60 cfm/person) also increased worker performance. These short-term effects on worker performance were demonstrated repeatedly, even at pollutant levels that increased SBS symptoms such as headache and poor concentration but had no measurable effects on the occupants' perception of air quality. These short-term effects have been validated recently in field intervention experiments in call centers in northern Europe and the tropics, and the effects were larger than those found in the laboratory.

The potential benefits of these improvements in workplace indoor air quality are substantial. Economic calculations based on this series of experiments indicate that benefits for improving indoor air quality beyond the minimum level acceptable to visitors would exceed improvement costs by a factor of about 60 (Wargocki and Djukanovic, 2003). Typical payback periods were estimated to be about two years. In another study, the economic benefits of reduced employee sick leave in offices can be achieved by increasing ventilation rates over the minimum recommended rate, as shown in epidemiological research in the U.S. (Milton *et al.*, 2000).

## 4. EXISTING REGULATIONS, GUIDELINES, AND PRACTICES

Despite the ubiquitous presence of toxic pollutants in the indoor environment, there are no government air quality regulations that are intended to protect the general public in residences, schools, or public buildings. Workplace regulations address indoor air quality, but they are designed for 8-hour exposures of healthy adults, and are not designed to be protective for longer periods nor for some of the more sensitive subgroups of the population, such as children and the elderly. Ambient air quality standards are focused on outdoor air quality, and are not designed to protect indoor air quality. Other regulations, such as California's Proposition 65, and AB 13, which prohibits cigarette smoking in workplaces, are applicable to indoor air quality only in a limited way and do not prevent indoor emissions and exposures.

There are a few examples of government regulations for emissions from specific sources of indoor pollutants that are intended to protect the general public in indoor environments. In addition, a variety of government agencies and private organizations have established voluntary guidelines and practices that can be applied to indoor environments to assist in the assessment and control of health hazards from air pollutants. The following sections summarize the pertinent regulations, guidelines, and practices for the following categories:

- Indoor and outdoor air quality in general.
- Emission limits for consumer products, appliances, and building materials.
- Building design.
- Building operation and maintenance.

### 4.1 STANDARDS AND REGULATIONS

#### 4.1.1 Workplace Air Quality Regulations

The California Occupational Safety and Health Program (Cal/OSHA) in the Department of Industrial Relations (DIR) has jurisdiction over most private and public employers and employees in California, with the exception of U.S. government employees. Cal/OSHA has regulatory authority to develop, promulgate, and enforce air pollutant exposure limits, ventilation regulations, and other standards for the workplace that directly impact indoor air quality. The California Occupational Safety and Health Standards Board is the unit within the Cal/OSHA program with authority to adopt standards and regulations to protect workers. Labor Code Section 144.6 requires the Standards Board to adopt standards that “most adequately assures, to the extent feasible, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to a hazard regulated by such standard for the period of his working life.”

Under CCR Title 8, Section 5155, the Standards Board sets permissible exposure limits (PELs) and other limits for airborne contaminants. The PELs legally apply to both industrial and non-industrial workplaces. However, they are not sufficient to prevent health impacts for all working individuals, such as individuals with pre-existing heart or respiratory disease, and they do not address possible impacts on those with asthma or reactions by sensitive individuals to low levels of chemicals. The Cal/OSHA PELs are 8-hour exposure limits designed to protect healthy working adults, and may be based in part on technological and economic feasibility considerations (non-health related criteria). These standards are not developed to protect infants, the elderly, or other sensitive groups who may frequent non-industrial workplaces (such as public buildings and retail establishments), nor are they intended to be protective for exposures greater than eight hours per day, five days a week. Additionally, PELs have not been

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developed for a number of known indoor air contaminants, and they are not designed to evaluate the health and comfort risks posed by the complex mixtures of pollutants found in modern buildings such as offices, schools, and homes. Generally, lower exposure limits would be necessary to protect the general population.

Both Cal/OSHA and others are aware that PELs do not fully address indoor air quality concerns in all workplaces. Cal/OSHA's Indoor Air Quality Policy and Procedure (C-48) states: "Most complaints about the quality of indoor air arise from employees who work in non-industrial environments...Approaches using traditional industrial hygiene techniques usually demonstrate compliance with 8 CCR section 5155 (PELs) despite the persistence of IAQ complaints from the building occupants." The Hazard Evaluation Section and Information Service (HESIS) within DHS also affirms that non-industrial workers experiencing indoor air quality problems are seldom exposed to contaminant levels approaching PELs.

Cal/OSHA also has regulations concerning the operation and maintenance of HVAC systems, and the control of moisture, vermin, and other sanitation concerns, as discussed later in Section 4.5. In addition, Section 3203, Illness and Injury Prevention Program, requires employers to have written plans for hazard identification, evaluation, and correction, for communication with employees, and for training. Other Cal/OSHA requirements reduce or prevent employee exposures to asbestos (Sections 5208, 1529), lead (Section 1532.1), and environmental tobacco smoke (Labor Code 6404.5, and Section 5148).

### 4.1.2 Ambient Air Quality Standards

National and State ambient air quality standards (AAQS), established by the U.S. EPA and the ARB, respectively, are developed to protect the general public from the harmful effects of "traditional pollutants" in outdoor air, for specified averaging times (exposure times). California's AAQS are often more protective than the national AAQS. Currently, the State AAQS are under review to ensure that they are protective of sensitive populations, especially infants and children (ARB/OEHHA, 2000). In the absence of indoor air quality standards or guidelines, the AAQS serve as useful guidelines for indoor air quality, because they are based on specified averaging times and incorporate a margin of safety. Outdoor standards for PM are often exceeded in indoor environments, and standards for CO, NO<sub>2</sub>, and ozone are sometimes exceeded. National and state AAQS are available at <http://www.arb.ca.gov/research/aaqs/aaqs.htm>.

### 4.1.3 Proposition 65

In 1986, California voters approved Proposition 65, the Safe Drinking Water and Toxic Enforcement Act of 1986, an initiative to address concerns about exposure to toxic chemicals. Proposition 65 requires the State to publish a list of chemicals known to cause cancer, birth defects, or other reproductive harm. The list includes approximately 750 chemicals, many of which are additives or ingredients in pesticides, common household products, food, drugs, dyes, solvents, building materials, and other sources found indoors. Businesses are required to provide a "clear and reasonable" warning when their products or actions may result in a release of chemicals above a specified threshold level, so that members of the public are aware they may be exposed to harmful chemicals. Warnings have evolved to include labeling of consumer products, posting signs at the workplace or on new housing, and publishing notices in a newspaper. OEHHA develops numerical guidance levels, known as "safe harbor" levels, for determining whether a warning is necessary. For potential carcinogens, the Proposition 65 "no significant risk level" is one excess case of cancer per 100,000 individuals exposed over a 70-year lifetime; for reproductive toxicants, the "no significant risk level" is one-thousandth of the no

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observable effect level (NOEL). Proposition 65 pollutants and safe harbor levels are available on the OEHHA website at <http://oehha.ca.gov/prop65.html>.

### 4.1.4 Tobacco Control in the Workplace and Public Buildings

Cigarette smoking is a major source of indoor pollution that is now prohibited in most public buildings in California. Passage of a statewide smoke-free workplace law in 1995 (AB 13, Friedman; Labor Code 6404.5, Section 5148) led to a reduction in smoking by the California population and eliminated smoking at nearly all California indoor workplaces, including restaurants, bars and gaming clubs. This statewide prohibition is primarily enforced at the local level. Cal/OSHA is required to respond to complaints of workplace cigarette smoke after the employer has been found guilty at the local level three times in the previous year. Prior to passage of the statewide law, numerous city and county ordinances had been implemented to restrict cigarette smoking.

The smoke-free workplace law contains fourteen exceptions to the smoking ban, each with additional explanations and stipulations. Exclusions include some hotel/motel guest rooms, hotel/motel lobbies, private meeting rooms, tobacco shops, truck cabs, warehouse facilities, gaming and bingo clubs, bars and taverns until January, 1997, theatrical productions, medical research sites, private residences, patient smoking areas, employee breakrooms, and small businesses. Despite the exclusions, the ban has been very successful in reducing worker exposure to cigarette smoke. In 1999, 93.4% of California's indoor workers reported working in a smoke-free environment, compared to only 45% in 1990 (Gilpin *et al.*, 2001).

The workplace prohibition of smoking has had far reaching benefits. The percentage of Californians with children under the age of 18 who do not allow smoking in the household has increased substantially. In 1994, 63.0% of Californians with children did not allow smoking in the house. By 2001, 77.9% did not allow it (Gilpin *et al.*, 2001). Fewer Californians are smoking as a result of this legislation and the DHS Tobacco Control Program. Smoking rates among California adults have declined from 26% in 1984 to 17% in 2001 (BRFSS, 2001). Californians who still smoke are smoking fewer cigarettes than they did in the past (Gilpin *et al.*, 2001).

### 4.1.5 Radon In Drinking Water

Radon levels in typical groundwater concentrations pose higher risks than those posed by the other drinking water contaminants that have been subjected to regulation (e.g., disinfection by-products). U.S. EPA was directed under the Safe Drinking Act (as amended in 1996) to attempt to regulate radon in drinking water. Though it has no authority to regulate indoor air radon, which is dominated by the soil gas infiltration, the U.S. EPA recognized that it would be far more cost effective to mitigate indoor air radon. The Safe Drinking Act gave U.S. EPA the latitude to allow higher concentrations of radon in drinking water if efforts were established to reduce indoor air radon to achieve risk reduction *equal to or greater* than the risk reduction that would be achieved by reducing the concentration of radon in drinking water (U.S. EPA, 1996).

The U.S. EPA is in the process of promulgating a maximum contaminant level (MCL) for radon, which is the allowable concentration based only on the contaminant risk in drinking water (U.S. EPA, 2000e). The proposed MCL is 300 pCi/l. An alternative MCL, 4000 pCi/l for states that had multimedia radon mitigation programs, and would allow utilities not to treat radon concentrations between the MCL and AMCL. Multimedia programs would aim to reduce indoor air radon risk, using a combination of approaches allowed in the regulations: public education; testing; training; technical assistance; remediation grants, loan or incentive programs; or other regulatory or non-

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regulatory measures. EPA (2000e) plans to make their multimedia program regulations effective in December 2005.

### 4.1.6 Reference Exposure Levels for Air Toxics

The Air Toxics “Hot Spots” Information and Assessment Act of 1987 (Assembly Bill 2588 as codified in Health and Safety Code Section 44300 *et seq.*) requires stationary sources of air pollutants (e.g., industrial plants) to report the types and quantities of substances their facilities routinely release into the air, ascertain health risks associated with the release, and notify nearby residents of significant risks. An amendment to the statute in 1992, SB 1731 (Calderon), requires OEHHA to prepare and adopt risk assessment guidelines, and requires facilities with a significant risk to prepare and implement risk reduction plans. Under this act, OEHHA develops acute and chronic reference exposure levels (RELs) as guidelines to prevent harm from toxic air pollution (<http://www.arb.ca.gov/toxics/toxics.htm>).

RELs are used by Cal/EPA agencies as indicators of potential adverse health effects other than cancer. RELs are generally based on the most sensitive adverse health effect reported in the medical and toxicological literature. They are designed to protect the most sensitive subgroups of the population by the inclusion of margins of safety. Because uncertainty factors are incorporated into the REL, an air concentration greater than the REL does not necessarily mean that the exposed public will suffer adverse health impacts. The methodology and studies used to develop the REL health standards are detailed in two documents available on the OEHHA website (<http://www.oehha.ca.gov/>): the *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part 1: The Determination of Acute Reference Exposure Levels for Airborne Toxicants*, March 1999 and the *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: The Determination of Chronic Reference Exposure Levels for Airborne Toxicants*, February 2003.

OEHHA has established chronic RELs for approximately 71 air pollutants. Chronic RELs are indicators of healthy versus potentially unsafe levels resulting from long-term exposure, exposure lasting at least 8% of a lifetime or 12 years and possibly longer. Chronic RELs are intended to indicate levels that will not cause adverse health effects in individuals with high susceptibility for chemical injury as well as identifiable sensitive subpopulations (high-risk individuals). However, chronic RELs may not necessarily be indicative of safe levels in hypersensitive individuals.

Acute RELs are indicators of healthy versus potentially unsafe levels of exposure for short periods of time, such as one hour. Because exposure is for a short time period, acute RELs are always higher than chronic RELs. OEHHA has established acute RELs for 51 chemicals (OEHHA, 2000a). OEHHA recommends that acute RELs be used to evaluate exposures that occur no more frequently than every two weeks in a given year.

In addition to providing an indication of healthful versus potentially unsafe levels outdoors near stationary sources, the acute and chronic RELs are also being used to identify healthful versus unhealthful pollutant levels in indoor air. Formaldehyde is a very common indoor air contaminant that has both cancer and non-cancer health effects. Because of the frequent use of RELs for indoor application, OEHHA developed an additional REL for formaldehyde, an interim REL (IREL) based on an 8-hour exposure period. The IREL established for formaldehyde is 27 ppb. The IREL identifies the level below which irritant effects such as eye, nose, and throat irritation would not be expected to occur during typical daytime (8-hour) occupancy of buildings. Other 8-hour IRELs are not yet available. Chronic RELs have been used in developing building material

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emission limits for use by DGS in specifying requirements for new state buildings and furnishings. These are known as Section 01350 requirements, and are discussed in detail in Section 4.3.3.2.

### 4.2 INDOOR AIR QUALITY GUIDELINES

Various governmental and private organizations issue guidelines to promote healthful indoor environments. Such guidelines cannot be enforced; compliance with the recommended levels is voluntary. Guidelines mentioned here are generally based on health or comfort endpoints.

#### 4.2.1 ARB Indoor Air Quality Guidelines

The ARB has published three indoor air quality guidelines – for formaldehyde, combustion pollutants, and chlorinated hydrocarbons. ARB's indoor air quality guidelines discuss the indoor sources and potential health effects of various pollutants, and provide information to the public on ways to limit or reduce their exposure to those pollutants. The guideline levels were developed in consultation with DHS and OEHHA, and are set to protect sensitive subgroups of the population. Because a number of the chemicals covered in the guidelines may cause cancer or other significant health problems, and no absolutely safe levels have been identified, recommendations are made to take action to prevent emissions of those chemicals in the home and to reduce exposure to the greatest extent feasible (ARB, 1991; ARB, 1994b; ARB, 2001b; ARB, 2004). Guidelines for traditional pollutants are based on the ambient air quality standards for California, because the basis of those standards is the health impacts seen at different air concentrations for specified exposure durations, which would be the same regardless of whether the individual is standing indoors or outdoors. An indoor air quality guideline for ozone is under development and is expected to be released in 2005.

#### 4.2.2 Air Quality Guidelines For Europe

"Air Quality Guidelines for Europe" were developed by the World Health Organization's (WHO, 2000) Regional Office for Europe, a specialized agency of the United Nations. The guideline value indicates a concentration of air pollutant below which no adverse effect to human health is expected, based on consideration of both carcinogenic and other health effects. They are intended to be applicable to both indoor and outdoor air exposures. Guideline values have been established for 38 non-carcinogenic compounds and some carcinogens for various exposure periods ranging from 15 minutes to one year. They address public health and are intended to be protective of the entire population. Uncertainty factors are used to extrapolate from animals to humans and from a small group of individuals to a large population. These factors are based on experience, wisdom, and judgement.

The latest edition of the WHO guidelines is more recent than the ARB indoor air quality guidelines. Some of the WHO guideline levels differ from the ARB guideline levels in part because they are based on more recent studies. For example, the current WHO NO<sub>2</sub> guideline is 100 ppb for 1 hour and 20 ppb for an annual average, while ARB's guideline is 250 ppb for 1 hour (a guideline for an annual average is not included). The ARB guideline coincides with the ARB ambient air quality standard for NO<sub>2</sub>, currently under review. If a new California standard is adopted, the guideline value may better align with the more recent WHO guideline.

Table 4.1. ARB Indoor Air Quality Guidelines

Pollutant	Measurement Period	Outdoor Air Quality Standards		ARB Recommended Maximum Indoor Levels
		ARB	U. S. EPA	
Formaldehyde (HCHO)	24-hour 8-hour	---	---	Lowest level feasible to reduce cancer  < 27 ppb to avoid acute irritant effects
Carbon Monoxide (CO)	8 hours	9 ppm	9 ppm	9 ppm
	1 hour	20 ppm	35 ppm	20 ppm
Nitrogen Dioxide (NO <sub>2</sub> )	24 hours	---	---	0.08 ppm avoid repeated high exposures
	1 hour	0.25 ppm	---	0.25 ppm
Particles – (PM10)*	24 hours	50 µg/m <sup>3</sup>	150 µg/m <sup>3</sup>	50 µg/m <sup>3</sup>
Particles – (PM2.5)*	24 hours	---	65 µg/m <sup>3</sup>	65 µg/m <sup>3</sup>
Polycyclic Aromatic Hydrocarbons (PAHs)	---	---	---	Lowest levels feasible to avoid cancer risk. Avoid or minimize exposure.
Chlorinated Hydrocarbons, e.g., chloroform Trichloroethylene <i>p</i> -dichlorobenzene methylene chloride perchloroethylene methyl chloroform	---	---	---	Lowest levels feasible to avoid cancer risk. Avoid or minimize personal exposures.

#### 4.2.3 DHS Mold Guidelines

The California Department of Health Services through the Division of Environmental and Occupational Disease Control (DEODC) is engaged in many types of activities regarding indoor mold. DEODC staff members provide current information about mold health effects and remediation recommendations through educational outreach to the general public, health care professionals, city and county government staff, and private environmental assessors and mold remediators. Staff members also conduct research to improve understanding of how indoor microbial growth affects human health, especially in sensitive groups such as pregnant women, children, and persons with asthma. DEODC Occupational Health Branch staff conducts health consultations in conjunction with Cal/OSHA to prevent harmful microbial exposures in California workplaces.

DHS Indoor Air Quality (IAQ) Program staff members have developed a website, [www.dhs.ca.gov/iaq](http://www.dhs.ca.gov/iaq), that provides information about mold-related health effects and voluntary guidelines for remediation of damp/moldy buildings through fact sheets and links to other good sources of mold-related information. Two fact sheets, "Mold in My Home: What do I do?" and "Mold in My School: What do I do", produced by DEODC staff have been used by other state agencies. The latter was selected by the National Clearinghouse for Educational Facilities to be



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distributed to teachers nationwide and included on the Clearinghouse website, [www.edfacilities.org](http://www.edfacilities.org). The DHS IAQ website also posts a “Listing of Consultants Offering IAQ Services in California” along with guidance for hiring IAQ consultants. This listing is a service allowing interested individuals to contact consultants in their geographic area. Because California does not license, certify, or endorse mold assessment or remediation professionals, individuals seeking assistance for moldy buildings are encouraged to use these and other reliable web resources to learn about this topic and find experienced consultants.

The DHS IAQ Program also provides staff to address inquiries from the public about mold and other indoor environmental problems through the Indoor Air Quality Telephone Assistance Hotline. Staff also organize and conduct meetings of the California Interagency Working Group on Indoor Air Quality (CIWG-IAQ), with representatives from State, Federal, local, and non-governmental agencies having IAQ oversight or interests. The CIWG-IAQ provides a unique forum for discussion, collaboration, and education on issues of indoor environmental quality, building engineering, and related health effects among the professionals working in the field.

The Toxic Mold Protection Act of 2001 (SB 732 Ortiz; California Health and Safety Code Sections 26100 et seq.) was enacted to address increasing concerns regarding health effects from exposure to indoor molds and to provide Californians with guidelines or standards for the safe and effective removal of molds from buildings. Among other tasks, this statute directs the California Department of Health Services (DHS) to determine the feasibility of identifying permissible exposure limits for indoor molds. DHS scientists have addressed this issue, and their conclusions are included in a report currently undergoing internal review. When approved for public release, this report will be posted to the DHS Indoor Air Quality website.

DEODC staff collaborates with other nationally recognized experts in developing guidance documents for mold assessment or remediation professionals, including:

- American Conference of Governmental Industrial Hygienists. *Bioaerosols: Assessment and Control*. ACGIH. Cincinnati, OH. 1999
- American Industrial Hygiene Association. *Field Guide for the Determination of Biological Contaminants in Environmental Samples*, 2<sup>nd</sup> Edition. In production. AIHA. Fairfax, VA.

### 4.2.4 American Society of Heating, Refrigerating, and Air Conditioning Engineers

The American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE) provides professional guidance on minimum building ventilation rates for human health and comfort. While not regulatory, ASHRAE Standards, especially Standard 62, *Ventilation for Acceptable Indoor Air Quality* (ASHRAE, 2003b), and Standard 55-2004 *Thermal Environmental Conditions for Human Occupancy* (ASHRAE, 2004) are important references for California’s ventilation standards and recommended comfort levels for nonresidential buildings. ASHRAE and other groups have also developed guidelines using indoor carbon dioxide concentrations as an indoor air quality indicator, ranging from about 800 to 1200 parts per million (ppm) as a “not to exceed” level.

### 4.2.5 U.S. EPA’s IAQ Tools For Schools Program

Although it does not provide indoor pollutant guideline levels, U.S. EPA’s *IAQ Tools for Schools Program* (<http://www.epa.gov/iaq/schools/>) provides schools with information they need to understand IAQ issues, prevent IAQ problems, and assure healthful indoor air quality. The



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program uses a team approach to school IEQ management and emphasizes staff and occupant training, communication, and improved routine operation and a district.

In California, U.S. EPA has trained more than 2000 individuals from districts throughout the state. Despite the outreach, awareness and use of the *IAQ Tools for Schools* program among California schools is still relatively low: about 11% of school districts use all or part of the program (ARB/DHS, 2003). U.S. EPA is partnering with organizations of school officials (e.g., ACSA, the Association of California School Administrators, and CASBO, the California Association of School Business Officers) to promote increased use of this program.

California Assembly Concurrent Resolution No. 75 (Chan), enrolled in June 2003, recognizes the significance of school indoor environments to the childhood asthma problem, and “encourages California school districts to implement the *Indoor Air Quality Tools for Schools Program* for the benefit of asthmatic children and for the health, well-being, learning, and productivity of the entire school population”.

### 4.3 EMISSION LIMITS

Prevention or reduction of emissions from pollutant sources is the preferred control technique for most pollutants. This section will discuss known regulations, guidelines, and practices for limiting emissions from consumer products, appliances, and building materials.

#### 4.3.1 Consumer Products

##### 4.3.1.1 Government Regulations For Consumer Products

###### Consumer Product Safety Commission (CPSC)

The federal Consumer Product Safety Commission (CPSC) has broad jurisdiction over consumer products, which are defined as any articles which are produced or distributed for sale to, or use by, consumers in or around the home or in schools, recreation areas, or other non-occupational settings. Certain products are exempted from CPSC authority, including pesticides, cosmetics, tobacco and cigarettes, food, drugs, automobiles, airplanes and firearms. In addition, houses and other buildings are specifically excluded by legislative history and judicial review. However, CPSC does have jurisdiction over home building materials that are sold or distributed as separate products to consumers.

CPSC has the authority under certain conditions to: (1) ban a product; (2) establish mandatory safety standards for products; (3) recall products for repair, replacement or refund; (4) mandate warning labels for products; and (5) cooperate with manufacturers in the development of voluntary product standards. The Federal Hazardous Substances Act (FHSA), enacted in 1960 and administered by CPSC requires labeling of “hazardous substances” if they are “intended, or packaged in a form suitable, for use in the household or by children”. A hazardous substance is further broadly defined as a “toxic” substance that may cause substantial personal injury or illnesses through reasonably foreseeable or customary use. In 1992, a supplemental definition was added to the Act to require appropriate labeling if a substance has chronic hazards of cancer, neurotoxicity, and developmental or reproductive toxicity.

CPSC has made a few notable contributions in areas related to IAQ regulation and research. Indoor pollutants regulated to some extent by CPSC through product bans or labeling requirements include asbestos, vinyl chloride, and combustion pollutants from appliances. However, voluntary product standards, rather than product bans and mandatory standards, are

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generally preferred by CPSC for several reasons. Voluntary standards take less time to develop and implement and are less likely to be challenged legally, so that public protection should occur sooner; they tend to be less intrusive, more responsive to technological change, and cheaper for all concerned.

The CPSC has focused on regulations for safety more than for health. For example, a policy on methylene chloride led to labeling regulations rather than a ban on its use in household products (CPSC, 1987a). The mandate of the California Department of Consumer Affairs (DCA) differs from that of the CPSC in that the DCA does not regulate consumer products directly, but rather regulates services provided to consumers and those who provide them, and addresses consumer complaints.

### ARB Consumer Products and Architectural Coating Programs

The focus of ARB's Consumer Products and Architectural Coating Programs is to reduce the amount of reactive volatile organic compounds (VOCs) that are emitted from the use of consumer products and architectural coatings. The driving force behind the state and local regulations developed under these programs is not reducing exposure to VOCs in indoor air, but limiting the release of VOCs that have the potential to react with other pollutants under sunlight to form ground-level ozone and particulate matter (PM<sub>10</sub>), the main ingredients in smog. As a result of these regulations, indoor air quality has improved in some situations where these products are used. For example, ARB prohibited the use of three TACs, perchloroethylene, methylene chloride, and trichloroethylene in 13 categories including general purpose degreasers, brake cleaners, all spray paints, all aerosol adhesives and adhesive removers. Additionally, antiperspirants and deodorants are not allowed to contain any compounds identified as TACs. Overall, these prohibitions have reduced TAC emissions in consumer products by over 10 tons per day (tpd). ARB performs an annual survey to determine remaining emissions of perchloroethylene and methylene chloride used in consumer products. The purpose of the survey is to determine if further mitigation measures are necessary to reduce potential health risks from outdoor exposures.

State air pollution regulations define "consumer product" as a chemically formulated product used by household and institutional consumers, including, but not limited to, detergents; cleaning compounds; polishes; floor finishes; cosmetics; personal care products; home, lawn, and garden products; disinfectants; sanitizers; aerosol paints; and automotive specialty products. Excluded from this definition are architectural coatings such as other paint products and furniture coatings. The VOC compositions of these excluded products are regulated by the local air pollution control and air quality management districts (districts), although the ARB provides considerable technical assistance to the districts and develops Architectural Coatings Suggested Control Measures (i.e., model rules) for adoption by the local districts.

The California Clean Air Act (CCAA) enacted by the Legislature in 1988 added Section 41712 to the California Health and Safety Code (HSC), which requires the ARB to adopt regulations to achieve the maximum feasible reduction in reactive organic compounds emitted by consumer products. In enacting Section 41712, the Legislature gave the ARB clear new authority to control emissions from consumer products, an area that had previously been subject to very few air pollution control regulations.

To date, ARB has adopted the following regulations to fulfill the requirements of the California Clean Air Act as it pertains to consumer products:

- Antiperspirants and Deodorants Regulation

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- Consumer Products Regulations
- Alternative Control Plan
- Aerosol Coating Products Regulation
- Hairspray Credit Program Regulation

Manufacturers are given flexibility in their approach to meeting the regulations. They can modify their product formulas to reduce VOC content or employ innovative products that may actually increase the amount of “active ingredients” and change the dispenser to lower the amount of VOC emitted per application. An alternative control plan allows manufacturers to average their emissions from noncomplying products with those from products that more than meet the standard. Variances provide temporary relief from the VOC limits in a product regulation if a company cannot comply for reasons beyond their control.

Although each consumer product may seem to be a small source of emissions, the cumulative use of these products by over 35 million Californians results in significant emissions. Consumer products accounted for approximately 267 tons per day (tpd) of VOC emissions in the year 2000, which comprised about 8% of the total man-made VOC emissions statewide. In 2005, without ARB’s actions to control VOC emissions in consumer products those emissions would exceed 400 tpd. Even with significant reductions from control measures adopted by ARB factored in, due to population growth, consumer products emissions are projected to total 260 tpd by 2010, and at that time make up about 12% of the VOC emissions projected to be emitted. Further reductions in VOC emissions from consumer products and other VOC sources are needed if ozone standards are to be achieved.

### 4.3.1.2 *Government Guidelines for Consumer Products*

A statewide sustainable building program can promote sustainable building concepts and challenge manufacturers to produce more environmentally friendly products. Recognizing this opportunity, Governor Davis issued Executive Order D-16-00 in August 2000. This Executive Order directed the Secretary of the State and Consumer Services Agency to develop a strategy for how the State could design, build, operate, and maintain buildings that are models of efficiency while providing healthy indoor environments. As part of that strategy, the Department of General Services (DGS) directed the development of specifications for environmentally preferable janitorial products for the cleaning and maintenance of state-owned buildings. Green Seal, a product certification organization, has recently developed a standard for cleaning materials (GS-37) that relies primarily on chemical content. The specification reduces emissions to the general work area, and reduces exposures to the individuals using the products. The specification is currently under revision to provide greater health protection. These specifications can result in reduced exposure to cleaning chemicals; however, the use of these products in state-owned buildings is voluntary.

### 4.3.2 *Appliances*

Indoor appliances that burn fuel (combustion appliances) or heat food but lack a venting system to remove combustion by-products, moisture, and odors, such as kerosene heaters, unvented gas logs, and unvented gas stoves, can be a major source of indoor air pollution. Wood stoves and fireplaces that burn gas or wood are nearly always vented by design, but they can also be a source of indoor air pollution due to leakage, re-entrainment of air pollutant emissions, or blockage of the flue. Building standards also require gas furnaces and gas water heaters to be vented, but the emissions of these appliances can sometimes leak or be drawn into a building due to blocked or malfunctioning vent systems.

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### 4.3.2.1 Government Regulations for Appliances

The design of combustion appliances is usually regulated through product standards, outdoor air pollution regulations, and indirectly through energy efficiency standards for appliances and buildings. The installation of combustion appliances in California is usually regulated through building standards that are enforced by local building officials, and through local air quality regulations that are implemented by local air pollution control districts. However, the performance testing or inspection of combustion appliances in California homes is not regulated unless the home is participating in a low-income weatherization (energy efficiency) program managed or approved by the State. These programs use a performance test method entitled the Combustion Appliance Safety protocol, or a modified version of it. Therefore, inspection of vented and unvented combustion appliances in California homes occurs mainly during home construction, major remodeling, and resale, but there are no emission or indoor air quality testing regulations for California homes unless the home is participating in certain weatherization programs.

#### Unvented Appliances

The sale of unvented combustion heaters designed for residential use, such as kerosene heaters, decorative gas logs and fireplaces, and gas-fired space heaters, has been banned in California since the 1980s (HSC Sec. 19881[a]). These appliances are still marketed for other uses such as workshops and barns, and are marketed by out-of-state sellers via mail order and the Internet. It is not clear how effective this regulation has been in preventing the residential use of unvented kerosene heaters, since 1 to 3% of California households use them as primary or secondary heat sources (Phillips *et al.*, 1990).

This regulation was amended in 1997 to allow the sale and indoor use of “natural gas fueled unvented decorative gas logs and fireplaces,” provided a series of steps were completed (SB 798, Haynes, HSC 19881[b-c]). The first step, the development of recommended standards by DHS and the Department of Housing and Community Development, was begun by funding a contractor study of the indoor air quality and health impacts of unvented gas logs. The contractor recommendations for unvented gas log standards included restrictions on emission rates for NO<sub>2</sub> and CO, with adjustments for different house volumes (Traynor, 1999). DHS recommended not setting a California standard for these appliances, and the manufacturers of unvented gas logs agreed with this approach. The sale of these devices is thus still banned under current regulations.

Cooking appliances that are unvented, especially gas-fired models, can also produce significant emissions of indoor air pollutants and water vapor, but these emissions are generally not regulated by any government agency. In addition, California regulations do not require cook stoves or ovens to have exhaust ventilation, nor do they limit the flow rate of the range hood to avoid excessive depressurization. The State Building Standard only requires that the range hood be able to supply the ventilation that is required by the stove manufacturer, but installation of a range hood or testing of the actual air flow rate for the installed hood is not required.

Residential building standards in some states have required that both electric and gas cook stoves have vented range hoods (State of Washington, 2003; MDC, 2004). In California, local building departments may adopt different building standards for the design and installation of appliances, as long as the local standards are at least equivalent to the state building standard code in performance, safety, and the protection of life and health (HSC 17951[d]2). For example, in its housing rehabilitation program, the City of Hayward (2002) requires range hoods to be exhausted to the outside.

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### Vented Appliances

Most of the 35 California air districts have rules governing outdoor emissions from vented gas appliances such as water heaters and residential central furnaces (ARB, 2003). Most of these rules require certification that natural gas-fired water heaters and central fan-type gas furnaces sold or installed in the district meet the 2002 emission limitations for NO<sub>x</sub> in South Coast Air Quality Management District's Rule 1121 (SCAQMD, 1999). This rule limits NO<sub>x</sub> emission to no more than 40 ng/J from gas water heaters and gas furnaces (the gas furnace rule does not apply to models for mobile homes). However, the SCAQMD emission limits for gas water heaters in homes will be lowered in 2005 to 10 ng/J of NO<sub>x</sub>. This reduction in NO<sub>x</sub> also reduces the potential magnitude of indoor pollutant exposure when water heater emissions enter a home due to backdrafting.

Outdoor emissions from wood burning devices have come under increasing control by local air pollution control districts in California. For example, in the Northern Sonoma County and San Luis Obispo County Air Pollution Control Districts, PM emissions from non-catalytic and catalytic wood fired appliances must be less than or equal to 7.5 g/hr and 4.1 g/hr, respectively. The districts also require U.S. EPA-Certified Phase II wood burning devices for new sales and installations (40 CFR, Part 60, Subpart AAA, Section 60.530 through 60.539b). State and federal regulations do not directly address the operation and maintenance of woodstoves to ensure that catalysts, door seals, and flue connections are operating properly.

Recently the San Joaquin Valley Unified Air Pollution Control District adopted fairly stringent regulations for fireplaces and woodstoves. These regulations: 1) ban the installation of fireplaces in most new residential development, 2) require U.S. EPA Phase II wood stoves in any sale, supply, transfer, and installation, and 3) require the removal of older woodstoves, with the option to install U.S. EPA Phase II or pellet-burning wood stoves, in any real estate sale or transfer (SJVUAPCD, 2003). The district can also curtail wood burning in the region when outdoor PM levels are predicted to exceed an Air Quality Index level of 150.

In the San Francisco Bay area, 24 cities have ordinances that prohibit conventional fireplaces in new construction. The mountain town of Truckee has a more aggressive policy that states that existing unapproved wood burning appliances must be removed from all properties by July 15, 2006.

California's energy efficiency standards for new home construction have provisions aimed to ensure proper venting and reduce indoor emissions. These standards require vented fireplaces, decorative gas appliances, and gas logs to be installed with closable doors over the opening, a combustion air intake for outside air, and a flue damper with a readily accessible control (CCR, Title 24, Pt. 6, Sec. 150[e]

[http://www.energy.ca.gov/title24/residential\\_manual/res\\_manual\\_chapter2.PDF](http://www.energy.ca.gov/title24/residential_manual/res_manual_chapter2.PDF)). In addition, gas-fired logs, lighters, or decorative appliances must have a flue damper that is permanently blocked open, as required by the California Mechanical Code or the manufacturer. Emissions from gas fireplaces are not regulated in California or the U.S. However, Canada has recently adopted an energy efficiency standard for both vented and unvented gas fireplaces. This standard is based on an industry consensus standard of the Canadian Gas Association (NRCAN, 2004a,b).

### Home Weatherization Programs and the Combustion Appliance Safety (CAS) Test

The California Department of Community Services Development (DCSD, 2003) manages the State Low-Income Home Weatherization Program, which provides insulation and air leak sealing for low-income homes. The program also requires completion of the Combustion

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Appliance Safety (CAS) test and appliance inspection before and after weatherization is completed, and cleaning and adjustment of the gas appliances to meet program specifications. The CAS test protocol was adopted to improve the health and safety of program participants, and to avoid unnecessary liability for contractors and government agencies. It is required by state weatherization programs in California and many other states.

The CAS test protocol specifications in California include the following:

- CO levels in the flue of the furnace and water heater cannot exceed 100 ppm, 100 ppm above the cooktop, or 225 ppm above the oven vent for both the oven and broiler burner. Cleaning, adjustment, and repair or replacement are required to bring the appliances within these limits.
- Clothes dryers must be vented outside with metal ductwork, and CO must not exceed 100 ppm in the exhaust vent.
- The flue draft pressures in furnaces and water heaters appliances must meet certain minimums
- House depressurization cannot exceed limits that can cause backdrafting of emissions from vented combustion appliances.
- Indoor CO cannot exceed 9 ppm in the room.
- An operable exhaust fan is required in the cooking area.

The California Public Utilities Commission has recently allowed investor-owned utilities to use a modified CAS for their low income weatherization programs (CPUC, 2003). For example, flue measurements of CO and installation of CO alarms are not required, while CO concentration in room air must be measured. If CO levels in a particular room (or rooms) are 10 ppm or more above nearby outdoor levels, then the utility may (but is not required to) conduct flue tests as a diagnostic tool to identify the source(s) of the problem. If the incremental indoor CO levels reach or exceed a 35 ppm threshold, the home must be ventilated, the occupants advised to evacuate, and the offending appliance made inoperable pending repair/replacement. CPUC will issue its final decision on testing requirements in early 2005.

### 4.3.2.2 *Government Guidelines for Appliances*

ARB's (1994b) Indoor Air Quality Guideline, "Combustion Pollutants in Your Home", recommends actions to reduce combustion pollutants in homes, including emissions from combustion appliances. These actions include removing unvented combustion sources from the home, using and maintaining vented combustion appliances properly, and ensuring adequate and balanced ventilation for the whole house. Also recommended are annual inspections and adjustments to combustion appliances, installation of CO alarms, and testing of combustion appliance safety. This guideline has been widely distributed in California and other states, especially through low-income weatherization groups, of which many are community-based organizations.

ARB (1997b) has also published a "Woodburning Handbook", which recommends measures for reducing emissions and exposures from residential woodburning. These measures include using a gas-fired furnace or gas fireplace insert instead, increasing the efficiency of the house and wood stove, and improving the operation and maintenance of the wood stove. This handbook has been widely distributed in California, mainly through local air pollution control districts.

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### 4.3.2.3 *Professional and Industry Guidelines and Practices for Appliances*

#### Combustion Appliances

Groups of high performance home builders and remodelers currently recommend using the CAS test protocol discussed above to prevent indoor air quality and moisture problems from combustion appliances. They also recommend providing exhaust ventilation for gas cooking appliances, and using sealed combustion or power-vented gas appliances that are immune to backdrafting. Examples of such building groups are the Energy Efficient Building Association (EEBA, 2003), the California Building Performance Contractor Association (CBPCA, 2003a,b), the U.S. Department of Energy's Build America program (BSC, 2003), and the National Association of Home Builders Green Building Program (NAHB, 2004).

Manufacturers of gas appliances currently test gas stoves, cooktops, furnaces, and other gas appliances using the industry-promulgated test standards and CO emission limits of the American National Standards Institute Standards Committee Z21/83. The ANSI standards for CO emissions from gas stoves can allow excessive levels of indoor air pollution to build up in homes when gas stoves are used for extended periods without adequate venting to the outside (Tsongas and Hager, 1994; Tsongas, 1995). ANSI is developing a test method for NO<sub>2</sub> emissions from gas stoves (Traynor, 1999).

For testing and assessing gas stoves installed in homes, "No industry standards or uniform government requirements currently exist..." (American Gas Association [AGA], 2003). Some, California utilities have been using the CAS test protocol to test existing installations (Hosler, 1998; see discussion of Weatherization programs above). For installing new stoves, the gas industry has adopted standards, but it is not clear how those standards prevent indoor air quality problems, how well they are incorporated into the State building standard, and how well contractors and homeowners comply with any such standards.

#### Carbon Monoxide Alarms

The standards organizations of the United States (Underwriter's Laboratory) and Canada (Canadian Standards Association International) have coordinated the writing of CO standards and product testing for CO alarms. The standards currently prohibit showing CO levels of less than 30 ppm on digital displays of alarms. The new standards also require the alarm to sound at higher levels of CO than with previous editions of the standard. New CO alarms will not sound at CO concentrations up to 70 ppm, concentrations that are significantly in excess of the health-based guidelines from ARB, WHO, and other groups. The reasoning behind these changes is to reduce calls to fire stations, utilities and emergency response teams when the levels of CO are not life-threatening. The CO alarm standards are not designed to protect the public from exposure to low-level, long-term exposures to CO, or from brief exposures to CO emissions from vehicle traffic or properly operating combustion appliances (UL, 2002).

Detectors with a digital display and a history option can provide the true CO concentrations in a house. A low-level display would be useful for people with existing respiratory problems or for those who like to spot evolving problems, rather than having to wait for the situation to become serious. Low-level CO detection products are becoming commercially available. They will not be certified to CSA or UL standards, as these standards currently prohibit low level displays.

#### Air Cleaners

The American Home Appliance Manufacturers (AHAM, 2004) has developed test protocols to certify the performance of room air cleaners in removing tobacco smoke particles, pollen, and dust. Testing is conducted by a third-party independent laboratory, and the test results are

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published. Certified air cleaners must also meet an ANSI/Underwriter Laboratories safety standard for ozone emissions of 100 ppb ozone. Unlike ozone generators, most models of room air cleaners are not designed to intentionally emit large quantities of ozone; however, ionizers and electrostatic precipitators can emit ozone as a by-product. The performance of air cleaners is also evaluated by consumer research organizations (Consumers Union, 2004a).

### Vacuum Cleaners

Vacuum cleaners can be an essential tool in controlling indoor pollutants that accumulate on interior surfaces. In recent years, vacuum cleaners have begun to include features such as high efficiency particle attenuation (HEPA) filters and electronic dust sensors that are advertised to improve the removal of allergens and dust from floors and furnishings (Consumers Union, 2003, 2004b).

To date, there are no industry consensus standards for testing and performance of vacuum cleaners. However, the Carpet and Rug Institute (CRI), discussed later in Section 4.3.3.3, has developed a voluntary “Green Label Vacuum Cleaner” program that includes tests for soil removal, dust containment, and carpet appearance retention. CRI provides a list of vacuum cleaner models that have passed these tests and received the Green Label on their website at <http://www.carpet-rug.com/>. CRI also encourages the proper use and maintenance of vacuum cleaners.

### 4.3.3 Building Materials

Conventional building materials such as vinyl flooring, carpet, paint, cabinets, and composite wood products can be strong indoor sources of chemical pollutants with a major impact on human health. In the absence of clear regulatory authority, governmental purchasing guidelines are being pursued to recommend low-emitting products in new construction. Also, certain segments of industry have initiated voluntary programs to reduce product emissions, and label their products accordingly.

#### *4.3.3.1 Government Regulations for Building Materials*

##### HUD Formaldehyde Emissions in Mobile Homes

The U.S. Department of Housing and Urban Development (HUD) has set limits for formaldehyde emissions from plywood and particleboard used in mobile homes. HUD has several mandated responsibilities that directly involve indoor air quality, although its basic mission is to provide adequate housing, promote community and economic development of urban areas, and eliminate discrimination in housing markets. In 1984, HUD established formaldehyde emission standards for plywood and particleboard used in mobile homes: test chamber concentrations are not to exceed 0.2 ppm and 0.3 ppm, respectively, to maintain indoor air concentrations of formaldehyde in mobile homes below 0.4 ppm [24 CFR 3280.309(a)]. CFR Title 24 Section 3280.309 also states that each new manufactured home shall have a health notice on formaldehyde emissions prominently displayed in the kitchen. The HUD mobile home standards preempt any existing or future standards of state or local governments that apply to the same aspect of mobile home performance (Sec. 604 [d]).

##### ARB Composite Wood Control Measures

An air toxic control measure (ATCM) is under development by the ARB to reduce the release of formaldehyde from composite wood products such as particleboard, medium density fiberboard, hardwood plywood, and composite veneer. The adhesive binding systems used to manufacture these composite products contain urea-formaldehyde resins, which release formaldehyde to the



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air. As discussed earlier, these resins can be a strong source of formaldehyde emissions (Kelly *et al.*, 1999; Hodgson *et al.*, 2002). It is estimated that formaldehyde emissions from composite building materials could exceed 400 tons per year in California (ARB, 2001c).

### 4.3.3.2 Government Guidelines for Building Materials

#### DHS Non-Binding Guidelines

*Reducing Occupant Exposure to Volatile Organic Compounds from Office Building Construction Materials: Non-Binding Guidelines* was developed by DHS (1996), in response to California legislation (Ch. 1229, Statutes of 1990). The guidelines present a simple technical approach for evaluating, selecting, and installing building materials in order to minimize occupant exposures to VOCs emitted from the materials in newly constructed or remodeled office buildings. The guidelines recommend a five-step approach to reducing exposure to VOCs:

1. Evaluate and select low-VOC impact building materials and products.
2. Pre-condition certain materials to minimize VOC emissions after installation.
3. Install building materials and products based on their VOC emission decay rates.
4. Ventilate a building during and after installation of new materials and products.
5. Delay occupancy until VOC concentrations have been reduced adequately.

The basic principles in these guidelines have been expanded more recently in manuals that describe “best practices” for assuring good indoor environmental quality, as described below

#### Sustainable Buildings and Section 01350

Several state agencies collaborate on task forces to develop guidelines for “green” buildings, also called sustainable buildings. Components of sustainable buildings include materials for interior surfaces and furnishings that are protective of indoor air quality and health, cost-effective, durable, recyclable, and contain recycled content. Under the direction of the Secretary of the State and Consumer Services Agency (SCSA), interagency task forces have been established for Sustainable Buildings (Executive Order D-16-00) and Environmentally Preferable Purchasing (AB 498, Chan, 2002; Public Contract Code Section 12400-12404), to develop and implement guidelines for construction, renovation, and operation of state buildings, and procurement of state purchases. Stakeholders include members from 40 governmental agencies including ARB, DHS, OEHHA, Integrated Waste Management Board (CIWMB), Department of General Services (DGS), Division of State Architect (DSA), and the California Energy Commission.

In 1999, the Legislature directed DGS to incorporate sustainable building measures into the design and construction of a \$392 million State office building complex in Sacramento known as the Capitol Area East End Complex. A product of the effort is *Special Environmental Requirements* (Section 01350; State of California, 2002), which provided protocols for testing of emissions of VOCs from building materials and furnishings to protect human health in state buildings (<http://ciwmb.ca.gov/GreenBuilding/Specs/Section01350/>). Manufacturers can have their products tested at independent labs using the test protocol developed for use at the Capitol Area East End Complex. Under Section 01350, primary emissions from a single material or product are modeled to estimate a room concentration; secondary emissions resulting from chemical reactions are not considered. The calculated room concentration cannot exceed one half the health-based chronic REL (developed by OEHHA for toxic air contaminants). Formaldehyde is an exception; emissions from a single product cannot exceed one half the interim 8-hour REL for formaldehyde, as discussed elsewhere in this document. The RELs have

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undergone extensive public comments and peer review by the State's Scientific Review Panel, so they are appropriate for use as an indoor guideline for this purpose.

Section 01350 has been incorporated into the DGS standard agreement for engineering and architectural services. It is also used in *Reference Specifications for Energy and Resource Efficiency* (CEC, 2004) and the *Collaborative for High Performance Schools (CHPS) Best Practices Manual* (CHPS, 2001). Section 01350 has been expanded to include additional details for broader applications such as schools, and products used in state buildings. Most recently, DHS has published their "Practice for Testing of VOCs from Building Materials Using Small Chambers" (<http://www.dhs.ca.gov/ehlb/IAQ/VOCS/Practice.htm>), which updates the indoor air quality portions of Section 01350.

### Collaborative For High Performance Schools

The Collaborative for High Performance Schools (CHPS) is a California consortium of public agencies and energy utilities working to facilitate the design and construction of "high performance" schools. These are school facilities that aim to be models of energy and resource efficiency, as well as healthy and comfortable settings supporting quality education. CHPS uses a whole-building design approach, as well as providing designers with specific guidance on component systems, that incorporates the best of current knowledge and technologies. CHPS developed their own grading criteria using a point system, similar to the U.S. Green Building Council's Leadership in Energy & Environmental Design (LEED™) rating system (U.S. Green Building Council, 2004).

The CHPS Criteria include prerequisites and optional measures for low- or no-emission building materials, furnishings, and cleaning practices. Construction practices to minimize indoor pollutant buildup or dispersion are also required. The building materials requirements are based on the Section 01350 specifications discussed above, with adaptations for classroom conditions.

### Environmentally Preferable Products for School Construction

As required under SB 373 (Chapter 926, Statutes of 2001), the Division of the State Architect (DSA, 2004) is developing a first of its kind database of environmentally preferable products (EPP) for use in school construction. SB 373 defines an environmentally preferable product as a product that promotes healthy indoor environments for children, and demonstrates the use of environmentally preferable materials and systems. When compared to other similar products with similar functions, an environmentally preferable product has some, or all, of the following characteristics relative to those similar products serving similar functions:

- Less hazardous to public health, safety and the environment.
- Consumes less energy in their manufacture or use.
- Contains more, or any amount of, recycled or post-consumer material content in their manufacture.
- Results in less potential waste.
- Results in less harm to indoor air quality.
- Consumes less water.
- Includes features, or is manufactured from materials, that promotes recycling or reuse of the product.

Development of the database is anticipated to include the following basic phases: research, criteria development, product screening, and database publishing. Products will be screened against a comprehensive set of environmental, health, and performance criteria. The indoor air

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quality criteria will be based on the requirements of Section 01350, discussed above. A research report looking at existing programs and resources pertinent to this project was completed in February, 2004 and posted on the project website (<http://www.eppbuildingproducts.org/>). Between April and July 2004, draft standards for composite panels, gypsum board, and fiber-based insulation were released for public comment and a series of public workshops was initiated. However, the Department of General Services has temporarily halted the project and is examining a potential change in the project approach, in order to ensure public participation. The criteria will be developed by working groups of state, federal, and local government, private and non-profit organizations and manufacturer representatives.

### 4.3.3.3 *Professional and Industry Guidelines and Practices for Building Materials*

Several building material industries have developed voluntary emission standards and programs to certify products that meet those standards. The most notable are the composite wood products industry and the carpet and rug industry. These programs have been successful in reducing emissions from their products over the last few decades.

The Composite Panel Association (CPA) is a professional organization for manufacturers of composite wood products. Composite wood products include pressed wood products such as plywood, particleboard, oriented strand board, and medium density fiberboard. The Association, founded in 1960, is dedicated to promoting the benefits of their products and producing products that meet a variety of rigid standards. Of particular note is the Grademark Certification Program that involves third-party certification of formaldehyde emission levels from wood panel products. This program uses the American National Standard Institute (ANSI) test procedures and limits for formaldehyde emissions from wood products.

The ANSI standard for particleboard and medium density fiberboard (MDF) establishes product specifications for dimensional criteria, physical and mechanical criteria, and formaldehyde emissions. In some cases, the industry formaldehyde emissions criteria are more stringent than those established by HUD for mobile homes. Standard test method ASTM E 1333-96, *Determining Formaldehyde Concentrations in Air and Emission Rates from Wood Products Using a Large Chamber*, is used to simulate and measure emissions during normal product use. Products from each manufacturer must pass this test to bear the CPA Formaldehyde Grademark Program stamp. The Hardwood Plywood and Veneer Association (HPVA) also uses the ASTM E 1333 Large Chamber test for product evaluation. For applications for which urea-formaldehyde resin compressed wood products are needed, ARB's guideline entitled *Formaldehyde in the Home* encourages the consumer to use composite wood products bearing the CPA or HPVA certification marks, because those marks confirm that the products meet relevant HUD and ANSI formaldehyde emission standards.

The Carpet and Rug Institute (CRI; <http://www.carpet-rug.com/>) initiated a voluntary testing program in 1994 as a result of the U.S. EPA's Carpet Policy Dialogue. Carpet samples are tested for chemical emissions by a third party laboratory according to ASTM D 5116 – *Guide for Small-Scale Environmental Chamber Determinations of Organic Emissions from Indoor Materials/Products*. For carpet, emissions limits were established at 0.5 mg/m<sup>2</sup> for TVOC; 0.05 mg/m<sup>2</sup> for 4-PC (4-phenylcyclohexene, the compound most associated with "new carpet odor"); 0.05 mg/m<sup>2</sup> for formaldehyde; and 0.4 mg/m<sup>2</sup> for styrene. Most, but not all, carpet manufacturers participate in the CRI test program. CRI also has emission standards and labeling programs for carpet cushion and adhesive. Products are tested quarterly to assure continued compliance with the test program requirements. Carpets that meet the emission test criteria bear a green and

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white CRI Indoor Air Quality Carpet Test Program logo in carpet showrooms so that consumers can identify the lower emitting carpet. Despite this labeling program, new carpet may still have a substantial “new carpet” odor, and extensive ventilation is recommended during installation and immediately after installation.

During the summer of 2004, at DHS’s urging, CRI further expanded their voluntary test program with the introduction of Green Label Plus ([http://www.carpet-rug.com/News/040614\\_GLP.cfm](http://www.carpet-rug.com/News/040614_GLP.cfm)), which includes some additional chemicals and emission limits from DHS’s Section 01350 test protocols. After a carpet product has been certified to meet the Green Label Plus criteria, it will be tested annually for the emission levels of 13 chemicals, and quarterly for emissions of total volatile organic chemicals (TVOC). The addition of some Section 01350 criteria is a major step toward further reducing carpet assembly emissions and assuring healthy indoor environments in newly carpeted areas, and addresses at least in part concerns that had continued regarding carpet assembly emissions. It is commendable that CRI has taken this action, and it is hoped that CRI will adopt any additional criteria identified in the future as necessary to assure healthful carpet and rug products.

The Greenguard Environmental Institute (GEI) has established performance-based, field validated standards to define products and materials with low chemical and particle emissions for use indoors. The standards establish certification procedures, including test methods, allowable emissions levels, product sample collection and handling, testing type and frequency, and program application processes and acceptance. Currently, over 20,000 different products are listed as certified under the Greenguard low emitting product standards (<http://www.greenguard.org>). Greenguard certification is a referenced standard incorporated in the U.S. Green Building Council’s LEED Program for commercial interiors, and is used by many municipalities and organizations across the U.S.

Greenguard requires products to be tested on an annual basis for over 2,000 individual chemicals including formaldehyde, measured carcinogens and reproductive toxins, as well as the sum of all measured chemicals. The modeled indoor concentrations of most individual VOCs detected must meet the concentration criteria of less than 1/10<sup>th</sup> of the threshold limit values (TLVs) established by the American Conference of Government Industrial Hygienists. However, as in the case of OSHA’s PELs for worker health and safety, using TLV fractions is not protective for non-industrial workplaces and residential environments. In fact, public health groups and ASHRAE (1981, 2003b) have abandoned this approach based on TLVs.

### 4.4 BUILDING DESIGN AND CONSTRUCTION STANDARDS AND GUIDELINES

Unlike some states, California does not have specific building design and construction standards to address IAQ. Standards for the design and construction of building ventilation systems and appliances in California are located mainly within Title 24 of the State’s building energy and ventilation regulations. These standards are promulgated by the California Energy Commission (Commission), and are enforced by local building officials, or in the case of public schools, by certified inspectors and the California Division of the State Architect.

For existing buildings, there are limited regulations or guidelines affecting indoor air quality. The Title 24 standards include requirements for substantial additions and alterations of buildings, including those for HVAC systems. Home weatherization programs operated by the State and some California utilities address the indoor emissions from combustion appliances and the building ventilation system. In addition, the Commission is beginning to consider energy efficiency programs for existing buildings.

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Guidelines for IAQ in building design and construction are available for the major building types, usually as an integral part of “green”, “healthy”, and “sustainable” building programs. In recent years, ARB and other State agencies have worked together, through the State Sustainable Building Task Force and the Collaborative for High Performance Schools, to develop IAQ measures to be used in sustainable buildings for the State and public schools. However, these IAQ measures have not yet been fully incorporated into all state building requirements, and are not currently required in non-State buildings, in schools, or in homes.

### 4.4.1 State Regulation of Design and Construction

The general mandate of the Commission is to ensure a reliable energy supply in a manner consistent with protecting the State's environment and enhancing its economy. The Commission develops and periodically updates energy efficiency standards for the design and construction of new buildings, appliances, and insulation materials. The Commission does not directly regulate the operation and maintenance of buildings and appliances, factors that are among the most important determinants of IAQ. However, some aspects of the Title 24 standards deal with HVAC controls systems, building operation modes, and providing building operation, maintenance, and design ventilation rate information to building owners and building managers upon occupancy (Sec. 10-03[b]). In addition, the Title 24 standards provide the basis for HVAC operation and maintenance standards developed and enforced by Cal/OSHA.

The Commission is required to routinely address indoor air quality impacts of its energy efficiency standards, under the authority of AB 4655 (Tanner; PRC 25402.8) and the California Environmental Quality Act. The Commission (CEC, 1994) prepared a report to the Legislature that assessed the indoor air quality impacts of its energy efficiency standards (PRC 25402.8). The Commission's standards and other activities pertinent to indoor air quality and building design and construction standards are summarized below.

#### *4.4.1.1 Design Ventilation Standards: Nonresidential, High-Rise Residential, And Hotel Buildings*

The California Energy Commission sets minimum ventilation standards for the design and construction of new buildings and building additions in nonresidential buildings. The standards also apply to high-rise residential buildings and hotels. These standards are published under CCR, Title 24, Part 6, and are required to be cost-effective. Local building officials enforce these State standards, but for public school buildings, the California Division of the State Architect (DSA, 2003) reviews the building plans and certifies building inspectors, thereby replacing the local building official function.

Currently, the 2001 edition of the Title 24 standards for nonresidential buildings require that newly constructed nonresidential buildings, and new HVAC systems in additions, be capable of supplying specific minimum ventilation rates for outdoor air. These minimum ventilation rates are typically met by a mechanical ventilation system that includes duct systems, control systems, and air filters. The standards require air balancing of airflows in the HVAC system, or airflow measurements, before occupancy in order to document that minimum ventilation rates are supplied.

The minimum ventilation rate is calculated by two alternative criteria. New HVAC equipment must be designed and installed to be capable of providing no less than the larger of the following:

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- The minimum ventilation rate based on the size of the building, expressed in cubic feet per minute (cfm) per square foot of conditioned building floor area. Rates required for different types of building use are shown in Table 4.2.
- The minimum ventilation rate based on the number of occupants: “15 cfm per person times the expected number of occupants.” The expected number of occupants may be specified by the designer. However, the expected number of occupants may not be assumed to be less than one half the maximum occupant load for existing purposes in Chapter 10 of the California Building Code.

These Title 24 standards also address the control of the HVAC system, which can markedly affect IAQ. The system and its controls must be capable of: 1) supplying the minimum ventilation rate at all times the building is occupied, and 2) providing one-hour pre-occupancy flushing at full occupancy ventilation rates, or at 3 air changes per hour or more. For spaces with high occupant density and at least 3,000 cfm capacity, such as auditoriums and large meeting rooms, the standards require “demand controlled ventilation” using a CO<sub>2</sub> sensor to achieve at least 0.15 cfm per square foot and maintain indoor CO<sub>2</sub> levels at or below 800 ppm. Such control systems have not been widely used in California, and it is not clear how well this requirement has been implemented. For the upcoming 2005 nonresidential Title 24 standards, the Commission required “acceptance testing” of demand control ventilation systems before occupancy, to ensure that the system performs as designed.

The Title 24 nonresidential standards also include an option for natural ventilation rather than mechanical ventilation. Natural ventilation by windows or roof openings is allowed if the openings are operable, accessible to the occupants, within 20 feet of the room’s outer edge, and sized to be greater than 5% of the conditioned floor area (this was recently increased to 25 feet for high-rise residential and hotel/motel guest rooms). Because there is no requirement for anyone to actually open the windows or to avoid nearby sources of outdoor pollutants or noise, the actual ventilation and IAQ provided under this option is highly variable, difficult to quantify, and difficult for the building owner or Cal/OSHA to verify.

### *4.4.1.2 Design Ventilation Standards: Low-Rise Residential Buildings*

Unlike nonresidential buildings, most homes in California do not have outdoor air provided by mechanical ventilation – outdoor air is provided instead via open windows and doors and by unintentional air leakage in the building shell and the HVAC ductwork. In 1978, the Commission adopted residential building design standards that required extensive caulking, weather-stripping, and sealing to reduce air infiltration in new low-rise homes. To avoid IAQ problems for very tight homes in a few climate zones of California, heat recovery ventilators (air-to-air heat exchangers) were required to provide outdoor air ventilation for certain home designs (Maeda, 2004), but very few builders used this option and the requirement was subsequently dropped.

More recent Title 24 standards for low-rise residential buildings require additional reduction of air leakage through the sealing of HVAC ductwork. The sealing of duct work not only reduces natural air infiltration, but also unintentional infiltration of pollutant sources from spaces adjacent to the home, such as crawlspaces, garages, vehicle traffic, and local woodburning. Compliance credit for additional reductions in building air leakage reduction can be obtained by wrapping the building shell with an infiltration barrier, but this measure is optional.

**Table 4.2. Minimum Design Ventilation Rates For Nonresidential, High-Rise Residential, and Hotel Buildings, 2001 Title 24 Standards<sup>1</sup>**

Type of Use	Cubic Feet per Minute per Square Foot of Conditioned Floor Area
Auto repair workshops	1.50
Barber shops	0.40
Bars, cocktail lounges, and casinos	1.50
Beauty shops	0.40
Coin-operated dry cleaning	0.30
Commercial dry cleaning	0.45
High-rise residential (5 stories or more)	Per CBC Section 1203 <sup>2</sup>
Hotel guest rooms (less than 500 sq. ft.)	30 cfm/guest room
Hotel guest rooms (500 sq. ft. or greater)	0.15
Retail stores	0.20
Smoking Lounges	1.50
All others	0.15

1. Adapted from CCR, Title 24, Part 6, Sec. 121(b) 2, <http://www.energy.ca.gov/title24/standards/index.html>.
2. Specifies minimum opening areas for operable windows, but does not require actual operation of window or maintenance of operability.

To help ensure adequate IAQ, the 2001 Title 24 standards require mechanical ventilation with outdoor air when the target infiltration is below a certain limit. Energy credits for “unusually tight” construction are not allowed unless: 1) air leakage is measured by an approved home energy rater using a blower door test method, 2) additional outdoor air is provided continuously by mechanical ventilation, and 3) combustion appliances such as furnaces and water heaters that use indoor air for combustion are not installed. The mechanical system may be either an independent system with its own fan, filter, and ductwork, or it may be integrated with a larger central air system. This approach is entitled “Compliance Through Quality Construction” ([http://www.energy.ca.gov/title24/residential\\_manual/res\\_manual\\_2001.PDF](http://www.energy.ca.gov/title24/residential_manual/res_manual_2001.PDF), p. 129).

A few other states have already required mechanical ventilation systems for new homes. For example, the State of Washington (2003) adopted the Ventilation and Indoor Air Quality Standard as part of its energy efficiency standards for new home design and construction. This standard requires continuous, whole-house ventilation, and exhaust ventilation by low-noise fans in the bathroom and kitchen. In addition, it requires that composite wood products be low-formaldehyde (exterior grade) and that a radon removal system be installed under the foundation. The State of Minnesota (MDC, 2004) has promulgated a state residential ventilation



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standard that requires continuous, whole-house ventilation, and exhaust ventilation by low-noise fans in the bathroom and kitchen.

### *4.4.1.3 Design and Performance Standards for Existing Buildings*

The Commission has begun to investigate strategies to achieve peak load reduction in existing buildings (mandated by AB 549, Longville). Such strategies may involve weatherization and duct sealing in existing buildings, which would affect the levels of indoor air pollutants produced by indoor pollutant sources such as combustion appliances and building materials and the levels of indoor moisture produced by human activities and other indoor sources. Strategies under consideration for non-residential buildings could affect and perhaps improve the operation of existing HVAC systems, e.g., retro-commissioning (performance testing) and the increased use of control systems to obtain optimum building system performance. The Commission's recommendations to the Legislature are expected in October 2005.

### *4.4.1.4 Standards for Home Weatherization Programs*

The California Department of Community Services Development administers the federal Low-Income Weatherization Assistance Program through trained and certified contractors (DCSD, 2003; U.S. DOE, 2003a,b). This no-fee program installs insulation, weather-stripping, caulking, duct sealant, furnaces, and air conditioners as needed in low-income homes, both renter- and owner-occupied. To avoid IAQ and ventilation problems, the program contractors use a standard protocol across the state to:

- Limit the tightening of the building shell. This minimum leakage rate is based on modeling of the amount of leakage typically needed to provide 0.35 air changes per hour when averaged over the heating season.
- Inspect and test combustion appliances, venting systems, and ductwork, as discussed above under Section 4.3.2.1, Emissions Limits: Appliances. Testing requirements include measurements of CO emissions, vent draft pressure, and building depressurization using the Combustion Appliance Safety (CAS) test.

## 4.4.2 Federal Regulations for Building Design and Construction

Federal standards do not currently exist for building design, except for new federal buildings, manufactured housing (mobile homes), and federal public housing. New federal buildings such as office buildings and residential buildings must meet federal design standards for energy efficient design, but federal policy has been to comply with local building standards where they are more stringent. Although California energy efficiency standards have long been more stringent than federal standards, the state and federal building ventilation standards are similar, except that new federal standards for new single family homes include mechanical ventilation. Standards for manufactured housing and HUD-financed public housing are discussed below.

### *4.4.2.1 Manufactured Housing*

Manufactured housing is regulated by HUD, which has developed requirements for low-formaldehyde plywood and particle board (but not medium density plywood), moisture control, exhaust venting, and fresh-air ventilation systems (HUD, 2002). These standards are enforced in California by the Manufactured Housing Section of the Department of Housing and Community Development. The formaldehyde emission requirements for wood products are discussed in Section 4.3.3.



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Earlier versions of the HUD standards gave homebuyers the option of the fresh-air ventilation system, but very few buyers in California selected that option. The current standard requires such a whole-house ventilation system and exhaust ventilation for kitchens and bathrooms. The whole-house ventilation system must provide 0.10 air changes per hour (ach), plus an assumed natural infiltration rate of 0.25 ach, for a total of 0.35 ach. However, a large study of newer manufactured homes in the Pacific Northwest found that many homes received much less than 0.35 ach, and that occupants did not use the ventilation systems as they are designed to be used (Lubliner and Gordon, 1990). In addition, some homes with wood fireplaces that have loose operable access doors have been observed to "backdraft" smoke into the homes, due to excess negative pressures caused by the HVAC equipment (Boe, 1999). The U.S. Department of Energy is funding research to improve the energy and indoor air quality performance of manufactured housing (Lubliner and Gordon, 1990).

### 4.4.2.2 *Public Housing*

Federal regulations for public housing financed by HUD require that the site characteristics, including the neighborhood and surrounding properties, undergo environmental review. HUD (1996) has provided a guide for this review, which addresses site hazards such as lead, PCBs from transformers, fuels, and solvents that could contaminate the homes during and after construction. At least three public housing sites in the U.S. have been built on known hazardous waste sites. Other housing built by the public or private sector may undergo a similar review through the California Environmental Quality Assurance (CEQA) review of environmental impacts, but the extent of the review for CEQA appears to be much less.

### 4.4.3 Professional and Industry Guidelines for Design and Construction

Several private and public sector organizations develop recommendations that serve as "consensus standards" or guidelines for achieving good indoor air quality through building design and construction. Many of these guidelines go beyond current regulatory requirements, but some are eventually incorporated into the regulations of government agencies. Concern has sometimes been expressed regarding the validity and effectiveness of such standards in protecting public health and welfare. However, although they may not meet all recognized needs, consensus standards serve an important function by providing guidelines for professionals such as building designers, managers and consultants, often long before government rule-making procedures could provide similar guidance. Government agency staff often actively participate in the development of such consensus standards by serving on standards-development committees of the major standards organizations.

ASHRAE has adopted a number of standards related to IAQ. These standards include those for ventilation and IAQ (Standard 62), air filter performance, thermal comfort, air-to-air heat exchanger performance, and building commissioning. These ASHRAE standards, especially Standard 62, have provided part of the basis for some building standards adopted by agencies such as HUD, CEC, and local governments. Thus, ASHRAE standards and their revisions have played a significant role in the previous and current State regulations for ventilation system design and energy-efficient building design as they relate to indoor air quality. Key guidelines from the public and private sector that address indoor air quality through building design, construction, and commissioning are summarized below.

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### 4.4.3.1 *Guidelines for Nonresidential Buildings*

ASHRAE Standard 62, Ventilation for Acceptable Indoor Air Quality, was first adopted in 1973. It was based on criteria for odor perception and indoor carbon dioxide concentrations, with a prerequisite that all outdoor and re-circulated air meet State or federal outdoor air quality standards. The Standard included minimum ventilation rates for various types of rooms in nonresidential buildings, and specified higher recommended ventilation rates for areas with smoking or other indoor sources of pollutants present. In 1981, ASHRAE Standard 62-1981 permitted lower ventilation rates in non-smoking areas, and included additional indoor air quality guidelines for several non-criteria pollutants such as formaldehyde and radon. The current version of Standard 62 for nonresidential buildings (ASHRAE, 2003a) specifies ventilation rates for outdoor air to adequately dilute pollutants emitted by occupants, occupant activities, and building materials, furnishings, and systems. For example, 15 cubic feet per minute (cfm) of outdoor air per person is required for most types of building occupants, and increased ventilation rates are specified for building areas with significant sources of indoor air pollution, such as bathrooms, printing and copying rooms, cooking areas, and garages. In addition, an appendix to the standard addresses the amount of ventilation required to reach recommended levels of specific indoor air pollutants. Standard 62-2001 is currently being modified under the continuous maintenance process; addenda to the standard are considered and undergo public review several times yearly.

### 4.4.3.2 *Guidelines for School Buildings*

Design guidelines for environmentally sustainable, healthy schools in California have been developed by the Collaborative for High Performance Schools (CHPS). CHPS Board Members include individuals from state agencies, utilities, and building professionals. CHPS (2001) has published the *Best Practices Manual* that describes such design features as selection of low-emitting materials, ventilation system specifications, and IAQ management plans. The *Manual* provides a self-rating certification system and the CHPS criteria for the indoor environment and related building topics. School designers have latitude to incorporate practices in the manner that best fits the district's application. CHPS also provides ongoing training for school facility staff, building designers, engineers, consultants, and manufacturers. At least ten school districts, including Los Angeles Unified School District and San Francisco Unified School District, have adopted CHPS Criteria for new school construction planning. A national version of CHPS has been adopted by a few other states recently. The U.S. EPA (2003b) has recently published "IAQ Design Tools for Schools," which is largely based on the CHPS *Best Practices Manual*.

CHPS recently added an Operations and Maintenance volume to the *Manual*, and plans further updates and additions in 2005. Recently, the U.S. Department of Energy adapted the CHPS *Best Practices Manual* Volume II (Design) for a national audience (U.S. DOE, 2003). Several school districts, notably Los Angeles Unified, have established policies to require all new facilities to meet the CHPS criteria, including those for emission testing of building materials. CHPS manuals are available free of charge on the Internet at <http://www.chps.net>.

### 4.4.3.3 *Guidelines for Residential Buildings*

For residences, ASHRAE Standard 62-73 and subsequent versions minimally addressed ventilation and IAQ. However, ASHRAE recently expanded Standard 62 to include ASHRAE Standard 62.2, which specifically addresses IAQ in low-rise residential buildings (Sherman, 2003a; ASHRAE 2003a). This standard now includes several IAQ features, such as whole-

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house mechanical ventilation with outdoor air, “local exhaust” fans in the kitchen and bathrooms, backdraft testing, and carbon monoxide alarms (Sherman 2003b). Ventilation rate requirements are 7.5 cfm/person, based on the need to remove pollutants generated by occupants’ indoor activities, plus 3 cfm/100 square feet of floor area, based on the need to remove pollutants generated by background or building-related sources. The standard also refers to Standard 62.1 for specific guidance in providing sufficient ventilation based on the pollutant emissions from the occupant activities and from the building materials and furnishings. This standard is for new home design, but can also be applied to existing homes. It is not a regulatory requirement in California, but it may help provide the basis for future State standards.

Professional home builder and public health groups in California have developed recommended practices for improving the performance of new and existing homes that take a “whole-building approach” to address building materials, ventilation, combustion safety, moisture control, thermal comfort, and energy efficiency. Examples of such groups are the Energy Efficient Building Association (EEBA, 2003), and the California Building Performance Contractors Association (CBPCA, 2003ab). In addition, the National Association of Home Builders (NAHB, 2004) is beginning to develop guidelines for “green” home building that consider the whole building and environmental concerns, and the Canadian Home Builders Association already has a green home building program (CHBA, 2004). The American Lung Association (ALA, 2004) has implemented the Health House Program, that includes builder guidelines, training, and educational information developed with a focus on preventing and reducing asthma and other indoor pollutant hazards. The HUD (1999) Healthy Neighborhood Program has funded several groups across the U.S. to use inspection and intervention to address asthma triggers, CO poisoning, lead poisoning, and other health and safety hazards for children in low-income populations. Numerous similar guidelines and programs have come into existence.

The recommendations from these groups generally include measures such as low-VOC building materials, whole house mechanical ventilation, exhaust fans for high-moisture areas such as bathrooms and kitchens, combustion appliance safety testing, and combustion appliances that are resistant to backdrafting and other venting problems. However, these approaches have not been widely used in California – only a few custom homes or demonstration projects have used these approaches in California so far.

### 4.4.3.4 *Building Commissioning Guidelines*

Building commissioning is the process of verifying and documenting that construction, operation, and maintenance of a building meet the design specifications of the project. Ideally, commissioning is conducted by a trained, third-party inspector, begins during the design phase of a project, and includes the development of operation and maintenance procedures and related training. Through commissioning, potential problems are detected early, HVAC systems are precisely tuned, change orders are reduced, building operators are trained, utility and other costs are reduced, and a healthful and comfortable workplace is produced (ODOE, 2004).

ASHRAE (1993, 1996) has published guidelines for commissioning HVAC and control systems in large buildings. The California Commissioning Collaborative (CCC, 2004), which includes government, utility and building services professionals, has been promoting building commissioning through incentives, training, research, and the development of model Requests for Proposals. Research funded by CCC members have found that in order to achieve building performance goals over the lifetime of the building, it is necessary to conduct enhanced training of building operators, to track building performance, and to conduct ongoing commissioning to meet current needs of building occupants (Friedman *et al.*, 2003).

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Building commissioning has proven to be very cost-effective in achieving reduced use of energy and other resources, while providing health and safety benefits through improved indoor air quality. The Commission included basic commissioning requirements in earlier Title 24 standards – air balance and flow testing of HVAC systems in nonresidential buildings (residential buildings do not have such requirements). The State of California has recently become more active in the building commissioning area.

- With guidance from the State Sustainable Building Task Force, DGS has begun to incorporate building commissioning in new projects for large office buildings. DGS is also beginning to “re-commission” existing state buildings.
- The Commission included HVAC commissioning requirements for HVAC control systems and major components in the 2001 Title 24 standards.

However, despite these and some related activities, formal and complete building commissioning is still relatively rare in California’s nonresidential buildings, and it is still in the methods development stage for residential applications (LBNL, 2003).

### 4.5 BUILDING OPERATION AND MAINTENANCE STANDARDS

#### 4.5.1 State Standards

The primary building operation and maintenance regulations in California are those of Cal/OSHA, which apply to most industrial and non-industrial workplaces. Non-industrial workplaces include beauty salons, hospitals, dry cleaners, medical laboratories, retail shops, copy shops, and many other workplaces that can at times have elevated levels of air pollutants associated with specific activities. School buildings are also subject to Cal/OSHA regulations, although school maintenance staff are not always aware that these workplace regulations apply. Residential buildings such as nursing and rest homes, daycare centers, and public housing used for special purposes also are subject to Cal/OSHA regulations.

The Cal/OSHA regulations most applicable to indoor air quality are Sections 5142 and 5143 (CCR, Title 8, <http://www.dir.ca.gov/samples/search/query.htm>). These regulations address the proper operation and maintenance of mechanical ventilation systems. Cal/OSHA enforcement of these standards mainly occurs when a worker files a complaint, and is therefore very limited. Other sections of Title 8, such as those addressing asbestos and lead, take a pro-active approach by requiring worker training, building assessment, and occupant notification.

##### 4.5.1.1 *Ventilation Regulations for Workplaces*

Employers are required to maintain and operate HVAC systems to provide at least the minimum quantity of outdoor air required by the State Building Code (Title 24) at the time the building permit was issued. Section 5142, *Mechanically Driven Heating, Ventilating and Air Conditioning (HVAC) Systems to Provide Minimum Building Ventilation*, was adopted in 1987 in response to a labor union petition. With limited exceptions, systems must be operated during working hours, unless the employer can document that the outdoor air requirements are being met by non-mechanical means. Employers must perform at least annual inspections, and correct problems found during those inspections. This section also includes record-keeping requirements; employees can obtain these records on request.

Section 5143, *General Requirements of Mechanical Ventilation Systems*, was adopted in 1976. This section requires annual performance measurement of ventilation systems that are used to

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prevent harmful exposures, such as local exhaust ventilation systems and laboratory hoods. This section requires contaminant-free make-up air (supply air), regular filter maintenance, and pressure gauge installation to indicate when filters must be cleaned or replaced. This section also has record-keeping requirements.

### 4.5.1.2 *Mold in Workplaces*

Cal/OSHA requires that workplaces be maintained in a sanitary condition. Section 3362 in CCR Title 8, *Sanitation – General Requirements*, was amended in 2002 to include new subsection (g) that requires employers to prevent and correct water intrusion, leakage or other uncontrolled accumulation because of the potential to cause mold growth. Other subsections require the employer to maintain the workplace in a clean, orderly, and sanitary condition; to prevent the entrance of insects, rodents or other vermin; to store putrescible wastes and garbage in covered cans; and to remove wastes as frequently as necessary to prevent harmful exposure.

### 4.5.1.3 *Construction-related Indoor Air Quality Investigations in Workplaces*

Many complaints to Cal/OSHA result from remodeling or other construction activities conducted in or adjacent to areas in which employees are working. In these situations, employers are required under Section 3203 to inspect the new operations, to evaluate hazards found, and to take measures to correct them in a timely manner. Section 5194 further requires employers to inform employees about hazardous substances in their work area, including substances being applied by contractors. In addition, the following Cal/OSHA sections require employers to control specific construction related hazards, such as asbestos, lead, and dusts.

Section 5208, *Asbestos*, was filed in 1996 and amended subsequently. It requires building and facility owners to determine the presence, location, and quantity of installed asbestos-containing materials (ACM) or presumed asbestos-containing materials (PACM), and to inform employers or employees in that building or facility of those results. Employers are required to provide that information to their employees. This section further requires training for employees who perform janitorial or maintenance activities in areas containing ACM or PACM, and the posting of labels or signs on installed ACM or PACM.

Section 1529, *Asbestos*, requires employers engaged in construction activities involving ACM or PACM to use certain control measures to protect employees in adjacent areas. Subsection (d) requires that asbestos contractors inform employers of employees in adjacent areas of those control measures, and it requires those employers to take measures to protect their employees from asbestos hazards and take steps on a daily basis to ensure that the control measures are adequately protecting their employees.

Section 1532.1, *Lead*, requires employers engaged in construction activities involving lead to use specified control measures, to establish a regulated area where exposures exceed the PEL, and to inform adjacent contractors of those control measures.

### 4.5.1.4 *Environmental Tobacco Smoke in Workplaces*

Environmental tobacco smoke historically has been strongly correlated with poor indoor air quality. Since 1995, California regulations (Labor Code 6404.5, Section 5148) prohibit smoking in most enclosed workplaces (also discussed in Section 4.1.5). This statewide prohibition is primarily enforced at the local level. Cal/OSHA is not required to respond to complaints until the employer has been found guilty at a local level three times in the previous year

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([http://www.dir.ca.gov/dosh/dosh\\_publications/smoking.html](http://www.dir.ca.gov/dosh/dosh_publications/smoking.html)). On May 29, 2003, in a Decision After Reconsideration (Robert D. Schultz and James A. Noll [OSHAB 01-125]), the Appeals Board held that the Division does not have authority to take action to enforce the Labor Code in the absence of a regulation promulgated by the Standards Board. Subsequently, in February 2004, the Standards Board adopted a workplace regulation for enforcing the smoking ban.

### 4.5.1.5 *Indoor Air Quality Enforcement Policy & Procedures for Workplaces*

Policy and Procedure C-48 (<http://www.dir.ca.gov/doshpol/p%26pc%2D48.htm>) describes Cal/OSHA enforcement procedures for indoor air quality investigations. In general, it defines “serious indoor air quality complaints” as those in which there is a known or suspected building-related illness, or a cluster of cancer cases or adverse birth outcomes. Non-specific complaints related to building IAQ are also considered serious when their magnitude, frequency, and their severity are above certain levels. “Non-serious indoor air quality complaints” are defined as those involving particles, HVAC systems, thermal stress, or symptoms that are not specific to a particular disease or pollutant. Investigations of building related illness might involve the Cal/OSHA medical unit, as well as support from state and local health agencies. Interview, inspection, monitoring, and citation procedures are also outlined.

### 4.5.1.6 *Standards for Nursing Homes and Public Housing*

Nursing homes, group homes, and community care facilities in California are regulated by the DHS Licensing and Certification Program (HSC 1265 et seq., HSC 1520 et seq.; HSC 1725 et seq.). The licensing requirements include provisions to protect the health and safety of clients. State inspectors conduct annual inspections or “surveys” and investigate complaints regarding the condition of the building, food preparation, and health care. They also make follow-up visits to assure that problems have been identified and corrected. However, it is not clear that IAQ is a high priority item for inspections unless there are several complaints by clients or an outbreak of a building related illness.

Public and Indian housing in California are funded by federal and state agencies, often through local public housing authorities and redevelopment agencies. Also, HUD provides funding for 5% of U.S. homeowners and renters through programs for public, assisted, and insured housing as well as housing financed through HUD’s formula for competitive grant programs (<http://www.hud.gov/news/release.cfm?content=pr02-105.cfm>). State agencies often provide additional funding. HUD has adopted regulations regarding the operation and maintenance of public and Indian housing that receives federal funds. Public housing authorities are required to annually set aside funds for building operation and maintenance. Some operation and maintenance activities are required to undergo environmental review (24CFR990.111). However, except for lead and asbestos contamination problems, public and Indian housing programs have not routinely addressed IAQ in their operation and maintenance programs.

### 4.5.2 *Guidelines for Building Operation and Maintenance*

Various guidelines for providing good indoor environmental quality through building operation and maintenance are available from the public and private sectors. Examples from California and national organizations are discussed below. Many other guidelines are available from other states and nations that have active programs to address indoor environmental quality.

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### 4.5.2.1 *IAQ Tools for Schools*

The U.S. EPA's *IAQ (Indoor Air Quality) Tools for Schools* Program is a program developed to help schools identify and prevent indoor air quality problems, using a team approach to school indoor environmental quality management. The program provides educational materials and tools for evaluating the impact of school maintenance functions and occupants' daily activities on indoor air quality. U.S. EPA makes their *IAQ Tools for Schools* action kits available at no cost, and has funded numerous training workshops, including many in California. Implementation of this program by school districts in California and other states has not taken a significant amount of staff or funding, but it has yielded substantial health and economic benefits (see Section 3, Costs of Indoor Air Pollution). As a prerequisite for obtaining state funding for school operation and maintenance, the State of Minnesota has required schools to adopt an IAQ management plan based on the *IAQ Tools For Schools* program.

Despite the outreach efforts for the *IAQ Tools For Schools* program, awareness and use of the program among California schools are still low: 35% of schools recently reported that they were familiar with the program, but only 11% of California schools reported that they use all or part of the program (ARB/DHS, 2003). These low levels may be due to a misperception regarding the level of effort required, although the program is adaptable to any level of resources.

### 4.5.2.2 *Large Building Air Quality Guidance*

U.S. EPA (1991) and the National Institute of Occupational Safety and Health have published "Building Air Quality: A Guide for Building and Facility Managers." This document describes the common principles of good facility management, provides background information on topics related to IAQ, and recommends strategies for problem identification and resolution in large buildings such as office buildings. The guidance emphasizes changing how building managers operate and maintain their building, not increasing the amount of work or cost of maintaining the building. The companion document, the "Building Air Quality Action Plan", is an easy-to-understand, 8-step implementation path for building owners and managers (U.S. EPA, 1998). The U.S. EPA (1997b) has also published guidelines for occupants of office guidelines, entitled "An Office Building Occupant's Guide to Indoor Air Quality". Most recently, U.S. EPA is offering I-BEAM, an interactive software program that integrates IAQ, energy efficiency, and building economics into a powerful management tool.

DHS (1995) has published guidance for occupants of large buildings with HVAC systems. Entitled "A 'Do-It-Yourself' Inspection of a Ventilation System," this document provides background information and guidance to help occupants identify potential causes of IAQ problems in their building, and to help them ask the right questions of building managers and investigators.

## 5. METHODS TO PREVENT AND REDUCE INDOOR AIR POLLUTION

There are five basic approaches to indoor pollution prevention and reduction of indoor air pollution. The most effective and reliable approach is usually source control. Source control avoids, removes, or reduces the sources of indoor pollution by using building materials, consumer products, and appliances that emit little or no air pollution. Ventilation also is necessary for healthful indoor air quality and comfort. It dilutes and removes indoor air pollutants to help reduce exposures, but not as effectively or reliably as source reduction. Proper operation and regular maintenance of buildings and their ventilation systems are key to preventing indoor air quality problems, especially over the long term. Public and professional education are also key to pollution prevention; they provide the awareness and understanding necessary to promote informed choices about preventing and controlling indoor air quality problems. Finally, air cleaning devices (air filters and air cleaners) can also be helpful in certain situations when used along with source control and ventilation.

An effective strategy for maintaining good indoor air quality must integrate all of these approaches. Many actions can be taken to prevent and reduce indoor air pollution and, in some cases, completely eliminate emissions of indoor air pollutants. The best approach will depend on the specific problem and will vary accordingly.

### 5.1 SOURCE CONTROL

Source control is the prevention or reduction of emissions at the source. It is the most effective and reliable approach to reducing indoor pollution because it keeps pollutants from entering and spreading throughout a building. It is the most reliable approach because it does not rely on building maintenance, or other human actions (ARB, 1989; NRC, 1981). Source control can be accomplished through source substitution, source removal, and source modification.

Source substitution involves using an alternative product that emits little or no pollutants of concern. For example, alternative building materials that emit little or no formaldehyde are generally available and can be used in place of a building material that emits formaldehyde.

Source removal involves eliminating the source from the building. For example, having family members smoke outdoors, and properly disposing of old cans of paint stored in a closet, removes these sources from the home. Source modification involves reducing the rate at which a pollutant is emitted into the indoor environment. Source modification could

**Minimizing indoor emissions is generally more effective than removing them after emission has occurred.**

*T.J. Kelly, Battelle, Indoor Air Quality Symposium:  
Risk Reduction in the 21<sup>st</sup> Century, Sacramento, May, 2000*

involve a change in design, formulation, or usage of a consumer product. For example, sealing all surfaces of a particleboard bookshelf can greatly reduce formaldehyde emissions (Kelly *et al.*, 1999; ATS, 1997; NRC, 1981). In addition, “green” cleaning products that have reduced levels of irritant and odorous compounds can reduce health and safety hazards for janitorial workers and occupants of buildings (OFEE, 2005; WRPPN, 2003).

Source control typically can be accomplished at the manufacturing level without direct impact on people’s activities and homes. Many consumer products, for example, have been reformulated by manufacturers to comply with ARB’s consumer product regulations to control emissions of



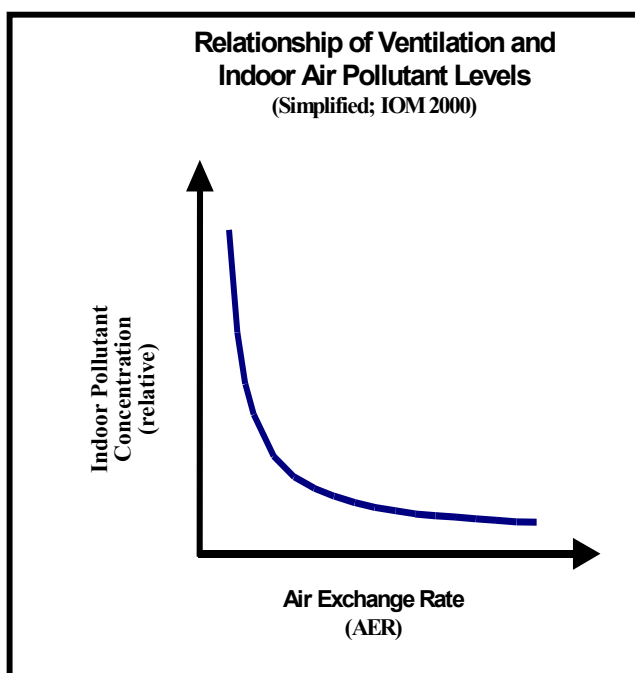
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reactive VOCs to the outdoors. Gas-fired furnaces, boilers, and water heaters have also been re-designed to comply with air quality management district limits for nitrogen oxides emitted to the outdoor air, but such regulations do not exist for emissions of nitrogen oxides to the indoor air, which can be substantial for unvented appliances such as gas stoves. Most such product changes have not been noticeable to consumers, or have had minimal impact on their choices and activities. Appliance standards could also be improved at the manufacturing stage to increase combustion efficiency, thereby lowering indoor and outdoor air emissions. Such standards might require reduced indoor nitrogen oxide emissions, automated local exhaust, and a clearly specified maintenance schedule.

Standard methods for testing emission rates are available for a number of indoor pollutant sources, such as consumer products, paints, carpets, and other building materials. Additional work is needed to develop test standards for other indoor pollutant sources, e.g., ozone emissions from air cleaners and office equipment, and combustion product emissions from gas stoves and fireplaces. More importantly, consumers, architects, building and interior designers, and building maintenance personnel need access to accurate and reliable information on the emissions of toxic and irritant compounds from products and appliances.

### 5.2 VENTILATION

Building ventilation is defined as air delivered to a space to dilute airborne contaminants. It serves many purposes. Ventilation removes and dilutes chemical contaminants and body effluents (such as carbon dioxide); maintains comfortable levels of temperature, humidity, and air movement; and helps remove excess moisture to prevent the growth of mold, bacteria, and dust mites. Thus, adequate and effective ventilation is necessary for acceptable indoor air quality, even when known air contaminants and sources are minimized. For instance, the air may have very low levels of measured air pollutants, but without ventilation, exhaled carbon dioxide, other body effluents, and non-measured air pollutants will build up, resulting in a stuffy, uncomfortable environment and potentially increased levels of toxic and irritant compounds. The figure illustrates the basic principle that indoor pollutants, body effluents, humidity levels, and so on are generally reduced as the number of air changes with outdoor air per hour (or air exchange rate) increases.



#### 5.2.1 Types of Ventilation

Ventilation can be "natural" or mechanical. Natural ventilation is the passive air movement through open doors or windows, and unintentional air infiltration through the cracks and gaps of the building envelope. Mechanical ventilation is the active movement of air through the building using fans to pull outdoor air in (make-up air), mix and circulate the air, and exhaust the indoor air to the outdoors.

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There are two types of mechanical ventilation – mixing and displacement. Mixing ventilation mixes “fresh” air and existing room air to uniformly dilute pollutants; this is the most common type of mechanical ventilation. Displacement ventilation is the introduction of “fresh” air so that the air around the occupants (in their breathing zone) is separated from the polluting sources. Displacement ventilation requires precision design and operation (Liddament, 2000). To operate efficiently and control air flows, mechanical ventilation systems require a relatively airtight building, hence, energy efficient builders commonly follow the principle of “build tight and ventilate right” (ESB, 1995).

Localized mechanical ventilation is often used to remove pollutants and increase ventilation in special use areas. For example, ducted exhaust fans are useful in bathrooms, laundry rooms, and kitchens to control odors, excess moisture, and potential mold growth. Local exhaust ventilation can be very effective when airflow is sufficient and exhaust fans are not too noisy. The use of exhaust hoods ducted to the outdoors is especially critical when using gas stoves or ovens, in order to remove the emissions from the appliances and those generated during cooking, which can be substantial.

For ventilating entire buildings, appliances such as heating, ventilation, and air conditioning (HVAC) systems are commonly used to provide mechanical ventilation, along with space heating and cooling. In California, these systems are used in most nonresidential buildings such as offices and schools, and usually include at least some minimal level of particle filtration for outdoor air. However, most residential buildings in California rely on natural ventilation for ventilating the whole building. California homes usually have a central air system, but rarely is it designed to bring in outdoor air or to run continuously when a building is occupied. Consequently, these residential systems mainly use recirculated air. They may have a small percentage of unfiltered outdoor air coming from unintentional leakage pathways from the garage, crawlspace, attic, or other areas.

Natural ventilation has limited effectiveness and reliability because it depends on building occupants to routinely open windows and doors. It also relies on indoor-outdoor temperature differences and wind speeds sufficient to force air through small cracks and other leakage points in a building, but these conditions are not consistently present in much of California. Natural ventilation does not allow for filtration of pollen and outdoor air pollutants, which can be substantially elevated in many parts of California. In addition, air flows from potentially contaminated areas such as garages and crawlspaces cannot be prevented by natural ventilation.

Despite its advantages, mechanical ventilation nonetheless has its limitations. It often does not provide adequate indoor air quality and comfort because of insufficient ventilation rates, poor air mixing within the building, uncontrolled air flows from contaminated areas, or poor quality of delivered air. These problems are usually due to improper design, operation, and maintenance of the HVAC system and local ventilation units (U.S. EPA, 1997b). For example, HVAC systems can produce excessive indoor noise, which lead occupants to frequently turn off the systems, as shown in the California Portable Classrooms Study (ARB/DHS, 2004). Building modifications may occur over the years without proper modifications to the HVAC system. Ventilation systems themselves also can be sources of indoor air pollutants, due to buildup of dust in ductwork and filters, and mold and bacterial growth in drain pans, cooling coils, filters, and ductwork. Inadequate training of building operators and insufficient maintenance funding also contribute to poor performance of HVAC systems.

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### 5.2.2 Ventilation, Health, and Comfort

The quantity and quality of the outdoor supply air plays an important role in assuring acceptable air quality in non-industrial buildings. A multi-disciplinary scientific consensus group recently reviewed over 100 peer-reviewed papers from the U.S. and Europe (Wargocki *et al.*, 2002). This group concluded that increased ventilation is strongly associated with improved comfort and improved health (SBS symptoms, inflammation, infections, asthma, allergy, short-term sick leave). Increased ventilation was also associated with improved productivity of office workers. For residential buildings, the authors concluded that ventilation rates above 0.5 air changes per hour in Nordic homes were found to reduce infestation of dust mites. However, air conditioning systems in non-industrial buildings with mechanical systems have been associated with the increased prevalence of SBS symptoms, indicating the need for better design, maintenance, and training of building operators (Wargocki *et al.*, 2002; Seppanen and Fisk, 2002).

The conclusions above from literature reviews are relevant to buildings in the U.S. and California. In a study of 41 U.S. office buildings, lower ventilation rates per occupant (as indicated by increased indoor CO<sub>2</sub> levels over outdoor levels) were significantly associated with increased SBS symptoms, increased absences, and reduced productivity among workers (Apte *et al.*, 2000). These associations held after adjusting the statistical model for several indoor air quality and occupant characteristics of the buildings. This study included some California office buildings.

### 5.2.3 Adequacy of Ventilation in California Buildings

Information on the quantity and quality of ventilation in California's public and commercial buildings is very limited. Neither Cal/OSHA nor other agencies routinely inspect or track ventilation rates in buildings with mechanical ventilation systems. The results of a recent large statewide survey of K-12 classrooms in California indicated that ventilation rates were inadequate for about 40% of the school day, and were severely inadequate about 10% of the time (ARB/DHS, 2004). Results from a small study of California buildings found that average air exchange rates (in air changes per hours) were 2.24 in 14 school buildings, 1.35 in 22 office buildings, and 1.35 in 13 retail buildings (Grot, 1995), and ranged from about 0.2 to 4.7 or larger. A substantial percentage of these buildings had minimum ventilation rates that did not meet applicable building energy and workplace regulations.

Information on ventilation rates in California's residential buildings is also limited. A few studies measured air exchange rates in homes of various ages, but all of the seasons and major regions of California have not been well represented. ARB (1998) summarized the available information, estimating that average air exchange rates (in air changes per hour) for California homes are 0.5 to 0.9 in the winter, and 0.7 to 2.8 in the summer, with spring and fall rates falling between the summer and winter values. The range of reported air exchange rates was very wide, as indicated by the large standard deviations and the lowest values near 0.1 air change per hour. A national study of air exchange rates from a database maintained by Brookhaven National Lab prior to 1993 indicated an overall mean air exchange rate for California and Arizona homes of 1.03 air changes per hour, and a 5<sup>th</sup> – 95<sup>th</sup> percentile range of 0.22 – 2.87 (Pandian *et al.*, 1993, 1998).

Newer California homes are likely to have lower air exchange rates, due to the increased “tightness” of new homes resulting from energy efficiency requirements such as the increased use of water vapor barriers on exterior walls, and the mandatory sealing of HVAC ductwork. As homes are made tighter, there is an increasing risk of degrading indoor air quality. To avoid this,

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the California Energy Commission specifically considers IAQ when setting state energy efficiency standards for buildings and appliances, as discussed in Chapter 4. However, very little data on ventilation rates and occupant ventilation practices are available for new homes in California. To assure that air quality in new California homes is healthful, the ARB and the California Energy Commission are jointly conducting a study to examine the adequacy of residential air exchange rates in new homes. It is especially important to avoid very low ventilation rates, which have been associated with the most severe impacts on IAQ and human health in some studies. The incremental cost of energy to increase ventilation rates to acceptable levels has been found to be minimal, especially when considering the avoided costs of impacts on human health and productivity.

### What is needed to improve ventilation in California buildings?

- ◆ Better commissioning and annual operational checks
- ◆ Quieter systems, especially for schools
- ◆ Control of building space pressures
- ◆ Use of higher efficiency air filters
- ◆ Better control of thermal comfort parameters (temperature and humidity)

*F.J. Offermann III, Indoor Environmental Engineering,  
Indoor Air Quality Symposium: Risk Reduction in the 21<sup>st</sup> Century,  
Sacramento, May, 2000*

To achieve the full benefits of building ventilation, additional work is needed to design mechanical ventilation systems that are quiet and easier to clean and maintain. A convenient, inexpensive but accurate method for assessing ventilation rates is necessary to facilitate routine testing of ventilation systems. Improved awareness of, and compliance with, state regulations for operation and maintenance of mechanical ventilation systems in schools and other workplaces are also needed.

Although it is necessary for adequate indoor air quality and comfort, ventilation is not a complete solution to indoor pollution. Ventilation consumes energy, and does not completely remove some pollutants, such as formaldehyde from building materials, that require years to off-gas. Ventilation reduces but does not completely eliminate near-source exposures when people use individual products in a building. Thus, it is generally preferable to remove highly toxic compounds from these products all together (ARB, 2000b).

## 5.3 BUILDING OPERATION AND MAINTENANCE

A well-operated building and regular maintenance is fundamental to providing good indoor air and a comfortable and productive indoor environment (Spengler, 2001; ATS, 1997). Basic building elements interact to affect the indoor environment. The building shell shelters the occupants from the elements, and at the same time may both emit and trap contaminants. The HVAC system conditions the air for comfort, but if not properly maintained it can add to the discomfort of occupants and become a source and dispersion method for mold, bacteria, dust, and chemical contaminants in the building (Clausen, 2004; Liddament, 2000; U.S. EPA, 1991).

Proper building operation and maintenance (O&M) for indoor air quality are comprised of several key elements. First, a written plan and maintenance schedule are needed to operate and maintain the building as originally intended and as appropriate for the specific building design, and to ensure that health and safety standards are met. The plan should address not only the routine maintenance after occupancy, but also the control of dust, fibers, mold, and chemical contaminants during building construction, repairs, and renovation. Next, the building should be commissioned after initial construction, after major renovations, and periodically

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throughout the life of the building. As discussed in Chapter 4, commissioning involves performance testing and inspection of the building to ensure that it performs as intended and that the various building systems work together properly. For example, commissioning helps verify the building's energy use, ventilation rates, thermal comfort levels, and use of specified building materials, air filters, and other environmental features. Commissioning also includes completion of the O&M plan and the initial training of O&M staff before the building is occupied. From the energy standpoint alone, commissioning by third party agents has been shown to be very cost-effective.

Another key element is training of O&M staff in the basic principles of indoor air quality and the proper methods to achieve good indoor air quality. Maintaining high quality indoor air requires a solid understanding of how buildings operate, the relationship between the HVAC equipment and indoor air quality, and how to achieve the design intent (Bearg, 1993). Regular cleaning with the proper equipment and methods can go far to eliminate some biological and dust contaminants. Effective cleaning, for example, involves using a vacuum cleaner with good filtration and efficiency, and allowing sufficient residence time of the vacuum head on the carpet or floor to assure good pick-up. Proper vacuuming can significantly reduce asthma and allergy triggers such as mold spores and animal dander in carpets, as well as pollutants that are often adsorbed onto floor dust, such as pesticides and PAHs. Similarly, cleaning properly with disinfectants and cleaning products can reduce bacteria and mold on surfaces and reduce exposure to potentially harmful bacteria. Training guidance is available from various sources mentioned throughout this report. Ongoing commissioning also is needed to assure that systems continue to perform as intended.

To implement a good O&M program, adequate funding is also essential. A reasonable funding level can be determined based on industry norms, experience in similar buildings, and on manufacturer recommendations for equipment repair and replacement. However, maintenance programs are often the first to be cut during a budget crisis, and the maintenance budget for California's public schools are especially strained (Patterson, 2000). Without adequate funding, maintenance programs fall further and further behind, thereby increasing the likelihood of major IAQ problem, structural damage, catastrophic failures, and litigation.

A good O&M program can not only help avoid worker productivity and health impacts from poor indoor air quality, but it can also help reduce the potential for litigation. Several lawsuits have been successfully litigated for mold cases in public buildings, commercial buildings, and homes. (Hirsch, 2005). Lawsuits over other IAQ problems such as CO poisoning, SBS, and pesticide poisoning have also been filed, but often end in out-of-court settlements. Litigation over IAQ has increased in recent years in the areas, especially in the area of mold damage (Facilities Network, 2005). This has led insurance companies to drop or limit their coverage for mold or water damage (Kats, 2003). It has also led to a proliferation of mold remediation companies that offer to inspect, test, and correct for mold problems. However, because there is no widely recognized licensing and training program for such companies, consumers and building owners are vulnerable to fraudulent practices and poor workmanship.

Litigation and liability risks do provide an incentive for better building design, operation, and maintenance, but they are not an efficient approach for society. Some IAQ cases are hard to prove because the toxic effects are chronic and delayed long after the initial exposures. In addition, litigation is very expensive, and individual homeowners and small businesses rarely have the resources to pursue litigation against a large company. Finally, increased litigation and liability may affect only business practices for certain IAQ problems such as mold, and may help the litigants but not the population in general.

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Building O&M is effective in preventing and controlling indoor air pollution, but only to the extent that proper training and adequate funding are provided. O&M can help achieve good indoor air quality if the indoor pollutant sources are controlled and the HVAC system is properly designed and commissioned, but this is not always the case.

In order to assure good O&M for buildings, additional work is needed to improve compliance with Cal/OSHA regulations for maintaining mechanical ventilation systems and preventing moisture intrusion in nonresidential buildings. For residential buildings, guidance is needed to improve the maintenance of the HVAC system, combustion appliances, and the building shell. Training of personnel in local health departments and building departments is also necessary to help them recognize and prevent IAQ problems in residential and nonresidential buildings and help enforce building and worker health standards.

### 5.4 PUBLIC AND PROFESSIONAL EDUCATION

People's choices and behavior have a major impact on their exposures to air pollution. Human activities are one of the key factors in determining the condition of the indoor environment. Cooking, the use of various consumer products, cigarette smoking, and other activities can result in immediate, significant releases of pollutants indoors which are immediately inhaled (ARB, 1987). Also, whether people choose to clean and maintain homes and buildings properly can have a major impact.

People are often not aware of the risks associated with indoor pollution and what they can do to protect their health. Many people assume incorrectly that any indoor air quality problem can be solved with an air cleaning device in their home or office, as witnessed by the recent boom in the sales of air cleaners. Sometimes activity pattern changes are needed, e.g., people must not smoke or must select different building products or consumer products to reduce their exposure and risk. Similarly, timely building maintenance and repair is critical to prevent mold development and accumulation of particles on surfaces. Even some building professionals are not aware of the impact of their lack of action on the building environment. Thus, increased awareness is a key step for reducing exposures to indoor air pollution (NRC, 1981; ATS, 1997).

Increasing people's awareness can be accomplished through public and professional education. However, public education will never be a complete solution. Some groups of the population cannot respond appropriately to take needed action. For example, children cannot read or understand all written information that is provided, elderly people living in group settings cannot control the products used in the facility, and low-income families may not be able to afford safer alternatives, even when they are fully aware of them.

Additionally, public education will not always reach all members of society. The many non-English speaking ethnic groups that enrich the California population pose an increased communication challenge. The California population contains a large number of people who speak a language other than English at home, as well as a large number of foreign-born individuals. According to the U.S. Census Bureau, the year 2000 census reported about 18% of the U.S. population 5 years and older spoke a language other than English at home. In California, that number is higher: about 40% of Californians speak a language other than English at home (DOF, 2002b). Also, the proportion of the California population that reported being foreign-born has increased from about 21% in 1990 to over 26% in 2000 (Malone *et al.*, 2003).

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Public education can change people's awareness and actions, but it does so slowly and requires a long-term commitment with adequate funding in order to reach the desired audiences and compete with commercial advertising. Because of these reasons, increased public education and information are not adequate as a sole approach to preventing indoor pollution.

The decisions of various persons and groups in the building, healthcare, and education professions can also help prevent and control indoor air pollution. Consumers and employers often rely on professionals to advise them on IAQ issues in their building, and perhaps to inspect and remediate their building. However, training for and awareness of IAQ is minimal in most professions involved with buildings and environmental health. Furthermore, there is no widely accepted training and certification program for IAQ professionals. Several groups offer certification as an indoor air quality expert, but the certification requirements are sometimes as minimal as completing a form or completing a one-day class. To change the awareness and actions of building owners and employers, existing training programs for building professions should be expanded, and adequate training on IAQ for professionals in the healthcare and education fields should be provided.

### **5.5 AIR CLEANING DEVICES**

Air cleaning devices are available in a variety of types and sizes. Most air cleaners remove particles, a few remove gases, and some perform both removal processes. Air cleaners can remove particles from the air using a mechanical or physical barrier, or through an electronic device. Mechanical air cleaners draw air through a filter with different sized pores that trap the particles. The most common types use a flat or pleated fibrous filter that can range from low to high efficiency at trapping particles. Electronic air cleaners are available in three basic types: electrostatic precipitators (ESPs), ionizers, and mechanical-electronic hybrids. ESPs use a small electrical charge to charge particles, which are then collected on oppositely charged plates as air is drawn through the device. Ionizers, or negative ion generators, also charge particles, causing them to stick to surfaces near the ionizer, such as the carpet and walls. Hybrid devices use both mechanical and electronic devices for pollutant removal. Air cleaners that remove gases and odors are less common, and relatively more expensive to purchase and maintain. Gaseous pollutants are typically trapped or destroyed as the air is drawn through materials such as activated charcoal or alumina coated with potassium permanganate. However, the filter material can become quickly overloaded and may need to be replaced often.

Air cleaning devices usually come as portable, stand-alone appliances, or as filters or cleaners in a central air system. Portable air cleaners are sometimes practical for rooms in existing homes where addition of a central air cleaner is too costly. Proper size, installation, and maintenance are critical for portable air cleaners to be effective. Test standards for particle removal by air cleaning devices have been developed by trade and engineering groups. The effectiveness of some portable air cleaners in removing particles is rated in terms of pollutant removal efficiency or clean air delivery rate (CADR), measured in cubic feet per minute (cfm). The CADR equals the airflow (cfm) multiplied by the efficiency of particle removal; a larger CADR is better. The CADR ratings are given separately for the removal of dust, pollen, and environmental tobacco smoke. Standards for gas removal have not been developed.

Portable air cleaning devices have limited utility, are often not very effective (ARB, 2000c; ATS, 1997; Consumers Union, 2003a), and can incur high-energy costs relative to the benefit gained. However, air cleaners may be useful for controlling airborne particles for some individuals with special sensitivities, such as those with asthma or allergies who use them in their bedrooms at night. Additionally, recent research has shown that in-duct ESPs can effectively reduce

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residential particles when the ESP is properly maintained (Howard-Reed *et al.*, 2003; Wallace *et al.*, 2004). Based on the limited scientific evidence that is currently available, the health benefits of air cleaners are not clear. Generally, it is more effective to prevent emissions rather than to try to remove them from the air once they are there.

For new homes or major remodels, "whole-house" or "fresh-air" ventilation systems that include some type of air cleaning device can be installed. Installed costs depend on the system size and the type of air cleaning device. Fresh-air ventilation systems are recommended in new, tightly built energy-efficient houses and for situations where the outdoor air is a major source of indoor pollution. To design and install any central system air cleaner, a company or contractor that is well experienced in designing and installing central filtration systems is important (ARB, 2005b).

### *Ozone Does Not Clean the Air!*

Some air cleaning devices emit ozone, either purposely or as a by-product of the particle-removal technology used in the device. Those that generate ozone by design are often called 'ozone generators'. ESPs, ionizers, and hybrid models often emit ozone as an unintentional by-product. While just a few studies have examined the health risk from the use of ionizers and electrostatic precipitators, research has shown that the use of ozone generators can result in harmful levels of indoor ozone.

Independent studies by the U.S. EPA, the Consumers Union, and others have shown that ozone generators can produce unsafe room levels of ozone up to several times higher than the state ambient air quality ozone standard of 90 ppb for one hour, reaching levels equal to a Stage One smog alert (200 ppb) (Mason *et al.*, 2000; Consumers Union 1992; Chen and Zhang, 2004). In a U.S. EPA study, ozone generators generated over 300 ppb in a test house within an hour or two of operation, and over 400 ppb in a chamber experiment (Mason *et al.*, 2000). In another study of a variety of air cleaner models tested in a chamber under realistic conditions, the ozone generators were the only models to exceed the OSHA 8-hour PEL for ozone of 100 ppb; levels again reached 300-400 ppb (Chen and Zhang, 2004). Personal air purifiers, which are typically small ozone-generating devices worn around the user's neck, can produce over 100 ppb in the user's breathing zone (Phillips *et al.*, 1999).

#### **Ozone-generating Air Cleaners**

"These machines are insidious. Marketed as a strong defense against indoor air pollution, they emit ozone, the same chemical that the ARB and USEPA (U.S. Environmental Protection Agency) have been trying to eliminate from our air for decades."

Barbara Riordan, interim ARB Chairperson. California Air Resources Board, Press Release 05-02, Sacramento, January 2005.

In addition to the potential harm posed by excessive ozone emissions, ozone generators also do not effectively destroy microbes, remove odor sources, or reduce indoor pollutants enough to provide any health benefits as manufacturers claim (Foarde *et al.*, 1997; Boeniger, 1995; Chen and Zhang, 2004). Ozone generators are often advertised as able to kill mold and bacteria even at "safe" ozone levels; however, ozone in the air must reach extremely hazardous levels (50-100 times the state standard) to effectively kill microbes (Foarde *et al.*, 1997). Ozone generators also are advertised as able to remove gaseous pollutants and odors, but ozone only reduces one group of VOCs, alkenes (a group that includes benzene). Additionally, ozone deadens the sense of smell. Most importantly, ozone reacts with other chemicals present indoors and leads



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to significant increases in the indoor levels of formaldehyde and other aldehydes, other VOCs, and ultrafine PM (Boeniger, 1995; Weschler, 2004). Some of these reaction products are listed as California toxic air contaminants, which can irritate the mucous membranes and respiratory tract or cause other health impacts.

Current market trends indicate that the use of ozone generators in California is widespread and growing. In an aggressive marketing campaign for ozone-generating devices, companies have targeted the most vulnerable populations, such as the elderly and persons with asthma or other respiratory diseases. Often, the advertisements used in the marketing of these devices make unsubstantiated claims and use misleading terms such as "super-oxygenated," "activated oxygen," or "trivalent oxygen," when referring to the ozone that is emitted.

State agencies have recommended that ozone-generating air cleaners not be used in the home (DHS, 1997; ALA, 1997; ARB, 2005). Presently, no state or federal agency has the direct regulatory authority to control emissions from these devices, although the Consumer Product Safety Commission has some authority to address products through a ban, required labeling, or other approaches. Efforts to restrict these devices through lawsuits, establishment of voluntary emission standards, cease and desist orders, and other means have all failed to restrict the sales and use of ozone generators.

## **6. PRIORITIZATION OF SOURCES AND POLLUTANTS BASED ON EXPOSURE AND ADVERSE IMPACTS**

Reduction of public exposure to the many indoor air pollutants is most effectively achieved by reducing pollution at the source. Because exposure is dependent on the presence and use of sources, pollutants are best prioritized for action by source categories. Mitigation approaches focused on source categories would be most effective for reducing pollutant exposure, and would generally be feasible to implement, although the feasibility would vary by specific source. Tables 6.1 and 6.2 suggest a prioritization scheme for implementation of mitigation measures, by source categories. The source categories have been ranked into two groups – high and medium priority – and are listed alphabetically within each ranking group.

The primary factors considered in prioritizing the source categories included the extent of the population's exposure to the sources and their emissions, the relative reduction in health impacts that could be achieved with further action beyond any already undertaken, ease of mitigation, trends in emissions from and use of source categories, and the extent of the gap in reducing exposure and risk from categories of indoor sources. A quantitative prioritization was not undertaken because such an effort is beyond the scope of this report. Such an effort would be an appropriate step prior to taking action under a comprehensive program to address indoor sources; a detailed prioritization based on quantified criteria would be needed. Additionally, the preliminary indoor air pollution cost estimates provided in this report were considered, but were not weighted heavily in the prioritization for several reasons. Most importantly, the cost estimates primarily reflect the availability of cost information and the length of time a given pollutant, such as ETS and radon, has been studied. Because of the lack of key cost data for most indoor pollutants, the cost estimates do not necessarily reflect the actual extent of exposure and risk in California, nor does it account for current information and trends in the scientific literature on exposure and risk. Thus, cost information was considered but was not a determining factor in the prioritization of sources.

Tables 6.1 and 6.2 also suggest potential approaches for mitigating the pollutants and sources listed. Emission reductions should be accomplished at the manufacturing, distribution, or construction stage. Alternatives or mitigation options are currently available for most of the sources listed. Emission limitations achieved at the manufacturing stage, such as reducing toxic contaminants in building materials, would be effectively invisible to the consumer and assure exposure reduction. Similarly, new appliance standards would reduce combustion emissions without reducing the range of product choices in the market place. With this approach, little or no change in individual behavior would be anticipated. As a result of public awareness and demand, there are an increasing number of "green" or low-emitting building materials, furnishings, and consumer products already available. Low-emitting carpets, no-formaldehyde furniture, and non-toxic cleaning products are currently available, and continue to grow in market share.

Finally, Tables 6.1 and 6.2 includes a column indicating whether direct authority exists at the state level to take the actions listed in column three. For most source categories, there is no state agency with clear, direct authority to take the mitigation actions indicated. In some categories, one or more agencies has limited authority to address a small portion of the sources included. For example, Cal/OSHA could impose product use restrictions or require other actions to reduce worker exposure to institutional cleaning product emissions. However, neither Cal/OSHA nor any other state agency has direct authority to restrict pollutant emissions from

Table 6.1. High Priority Source Categories for Mitigation: <sup>1</sup>

SOURCES OF POLLUTANTS <sup>2</sup> (listed alphabetically)	EXAMPLES OF POLLUTANTS <sup>3</sup> EMITTED	POTENTIAL APPROACH TO MITIGATION <sup>4</sup>	DIRECT STATE AUTHORITY TO TAKE IAQ MITIGATION ACTIONS
<b>Air Cleaners</b> (ozone-generating)	Ozone	Emission limitations	No
<b>Biological Contaminants</b> (mold, pollen, bacteria, viruses, house dust mites, cockroaches)	Particles, allergens, asthma triggers, toxins	Requirements for habitable spaces; require certification of mold assessors and mitigators	Limited
<b>Building Materials &amp; Furnishings</b> (particle board, plywood, paneling, flooring, caulk, adhesives, new carpet assembly, furniture)	Formaldehyde, acetaldehyde, benzene derivatives, acrylates, naphthalene, phenol, some other VOCs	Emission limitations, product use restrictions, market incentives	Limited (some indirect)
<b>Combustion Appliances</b> (gas & propane stoves, ovens, furnaces, heaters; woodstoves and fireplaces)	Carbon monoxide, nitrogen oxides, particles, soot, polycyclic aromatic hydrocarbons	Emission limitations, active exhaust ventilation, safety devices, product use restrictions, product re-design, improved venting	No
<b>Environmental Tobacco Smoke</b> (cigarettes, cigars)	Particles, polycyclic aromatic hydrocarbons, benzene, carbon monoxide, some other VOCs	Focused parent education; reduce smoking in homes and vehicles	Yes, workplaces  No, private homes and vehicles
<b>Radon</b> (soil, rock, ground water, building materials containing radium)	Radionuclides, radon gas	Screening measurements, building codes	Limited

1. Individual sources may be higher or lower than the source category ranking.
2. All of the examples of pollutant sources may not emit all of the pollutants listed in the corresponding box in column two.
3. Air pollutants may be identified as toxic air contaminants (TACs) by the California Air Resources Board, and/or identified as Proposition 65 chemicals; or, criteria (traditional) air pollutants.
4. Public education, economic incentives, and non-regulatory approaches should also be used where appropriate. The actual approach taken would be determined after extensive discussions with the relevant industries, in consideration of costs, feasibility, and effectiveness.

cleaning products for the purpose of reducing indoor air concentrations and exposures. Also, for most categories, any related authority is usually indirect – the authority is not focused on reducing indoor pollution to protect public health, but rather is a mitigation measure to reduce

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impacts of actions taken under the primary authority, or is incidental to the primary regulatory activities. For example, one mission of the Energy Commission is to improve energy efficiency in California's buildings. The Commission has been attentive to indoor air quality impacts that might result from their regulatory actions, and includes provisions to prevent adverse impacts on indoor air that might result. However, the Commission does not have authority to develop a regulation to address indoor air quality outside of their energy-related missions. Finally, while the ARB has regulatory authority over some indoor sources such as consumer products, that authority is targeted toward actions to reduce the impacts on outdoor air quality. Some such actions taken by the ARB have had positive impacts on indoor air quality as well, but those impacts are either incidental to the actions taken for the purpose of reducing or preventing outdoor air pollution, or are mitigation measures designed to reduce adverse environmental impacts from ARB regulations.

The specific rationale for the ranking of each category is discussed below. Note that the prioritization is a ranking of the overall group of sources included in a given category: some individual sources within the group may have a higher or lower priority. A more detailed assessment would be needed to prioritize specific products within these larger categories.

### 6.1 HIGH RANKED SOURCE CATEGORIES

- **Air cleaning devices or "air purifiers" that generate ozone** should not be used in occupied places, and their ozone emissions should be limited. Some devices marketed as air cleaners purposely release ozone, which can directly harm sensitive occupants. These air cleaners should not be used in occupied spaces because they can produce potentially harmful levels of indoor ozone, up to several times the state outdoor standard level and even a Stage 1 smog alert level. Additionally, they are ineffective at safe levels. Safe, effective alternatives are available in the marketplace. Additionally, ionizers and electrostatic precipitators, two other types of indoor air cleaners, emit ozone to varying degrees as a by-product of their function. These devices are effective at removing particles, but poor maintenance and old age can result in increased ozone emissions. Ozone emissions from these devices should also be limited to assure that indoor concentrations remain well below concentrations that may harm sensitive individuals. Consumers often seek advice about the type of air cleaner they should buy to meet their specific needs, but there are few criteria available to guide consumers in their purchase. Emission limitations are needed for these devices, which are currently unregulated.

A program to reduce the use of high-ozone-emitting devices and to educate the public should be undertaken. Such a program might include:

- a) Development of public and professional guidance materials for indoor ozone.
  - b) A public and professional outreach program to alert them to the potential harm of ozone generators and safe alternatives. Such a program should target medical and dental professionals, building managers, school administrators and facility managers, senior citizen organizations, public health entities, environmental organizations and others.
  - c) Development of test protocols for air cleaners, to include methods for testing ozone emissions as well as for effectiveness of cleaning the air.
  - d) Meetings with manufacturers and their professional organizations to facilitate actions to address this issue.
- **Biological contaminants** are a group of both sources and pollutants. Some mold and bacteria can emit chemical toxins, but for the most part the organism or its parts actually

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cause the effect. Biologicals are a high priority because of their ubiquitous presence and their widespread health and fiscal effects. Animal dander, pollen, house dust mites, and cockroaches cause millions of sensitive individuals to experience allergy symptoms and asthma attacks. Indoor mold has been an increasing problem in recent years, costing substantial sums of money for remediation and lawsuit settlements. Bacteria such as *Legionella* cause both serious illness (for example, Legionnaire's disease) and shorter-lived disease (for example, Pontiac Fever). While not emitted into the indoor environment per se, infectious disease transmission is increased in indoor environments with crowded or dirty conditions and insufficient outdoor air flow. Improved methods are needed to objectively and efficiently identify and quantify dampness and microbiological growth in building, especially within wall cavities or other areas that are difficult to access. Additional research is needed to develop such methods.

The mitigation approaches for biologicals are varied. Mitigation actions for mold and some other biologicals might include required annual inspections and remediation in public buildings, group homes, and rental units, and in private homes at the time of sale. Certification requirements for mold assessors and remediators would help assure the quality of inspections and remediation.

- **Building materials and furnishings** are a high priority for mitigation because they often emit multiple toxic air pollutants, especially when new, and have a high loading level in indoor environments, resulting in high exposure levels for occupants. Additionally, a substantial percent of the population is exposed to such emissions due to the continued high rate of new building construction in California and the increasing number of home renovations undertaken by homeowners. Emission limits for pollutants emitted from building materials and furnishings (formaldehyde being the most predominant) would benefit all indoor environments and has potential for significant health benefits due to reduced incidence of asthma exacerbation, cancer, and eye, nose and throat irritation.

Low-emitting alternatives are available. Non-wood alternatives and composite wood products made with phenol-formaldehyde resin or methyl diisocyanate (MDI) have much lower formaldehyde emissions than composite wood products made with urea-formaldehyde resin, and could be substituted for some applications. In cabinets and furniture, all surfaces of these products can be coated or laminated to substantially reduce formaldehyde emissions. Building materials are currently available that meet Section 01350 emission requirements for formaldehyde and other chemicals of concern (Alevantis, 2003). Low-emitting modular office furniture was used in the Capitol Area East End Complex, and could be used in all future state and private offices. Numerous governmental groups focused on sustainable building and environmentally preferable purchasing are compiling lists of acceptable products that have a low impact on indoor environments. For example, a list of products for use in school construction projects that meet Section 01350 requirements is available at [http://www.chps.net/manual/lem\\_overvw.htm](http://www.chps.net/manual/lem_overvw.htm). DSA is working on a similar list. These low- and no-emitting alternatives are available and should be required in public buildings, group homes, schools, and other buildings.

- **Combustion appliances** are also a high priority for mitigation. They emit carbon monoxide, nitrogen dioxide, polycyclic aromatic hydrocarbons, particles, and other pollutants, depending on the appliance fuel, status of adjustment and maintenance, and other factors. These pollutants can have severe acute health effects including respiratory effects and exacerbation of asthma, and contribute to cancer risk. Reduced exposure to pollutants from gas and propane appliances, whether it be through emission limitations, active exhaust

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ventilation, or both, could have immediate widespread benefits for occupants in environments with such appliances. Precedence for mitigation of appliance emissions has been set in the low-income weatherization program (DCSD, 2003). Guidelines for carbon monoxide levels and venting requirements in this program can be expanded to statewide regulations.

Statewide measures to reduce emissions from woodstoves and fireplaces both indoors and outdoors also are highly desirable. Such measures could have a major impact on improving both community-wide indoor and outdoor air quality in many areas of the state. Emission limitations, product re-design, product use restrictions, and improved venting can be used for reducing this type of pollution. A number of local government entities have recently approved regulations restricting the use of woodstoves and fireplaces: in the San Francisco Bay area, 24 cities have ordinances that prohibit conventional fireplaces in new construction. The mountain town of Truckee has a more aggressive policy that requires that existing unapproved wood burning appliances be removed by July 15, 2006. The San Joaquin Valley ([www.valleyair.org/Burn\\_Programs](http://www.valleyair.org/Burn_Programs)) implemented a daily advisory for restrictions on residential fireplace or wood stove use on January 1, 2004. Woodsmoke especially impacts those with asthma and other respiratory disease.

- **Environmental tobacco smoke** has been greatly reduced in California, primarily due to legislation that bans smoking at the workplace. However, ETS remains the number one cause of lung cancer, and should be reduced to the greatest extent feasible. Children's exposures remain a special concern, because they can be highly exposed when smoking occurs in their home or in vehicles driven by family or friends who smoke. Actions to reduce children's exposure, such as an increased focus of public education on smoking parents, and reduction of smoking inside vehicles and homes with children, remain a high priority. Additionally, some outdoor and commercial building (casino) exposures continue to occur, with potential for exposure reduction.
- **Radon** gas and its radionuclide precursors enter indoor environments largely from uranium-bearing soil or rock under and near the building. Despite the preliminary high lung cancer risk from radon estimated in this report for California, the lung cancer risk from radon is essentially inseparable from that of smoking, making reduced exposure to tobacco smoke the most effective mitigation approach. Radon levels in California are relatively low, except in a few less populated areas such as the Sierra Nevada foothills and the Ventura Mountains, and recent measurements in the Sierras indicates that statewide radon levels may be lower than estimates derived from measurements obtained in the 1980s. Mitigation approaches usually include depressurizing the basement, crawl space, or sub-slab region to reduce infiltration. However, mitigation is not recommended until adequate testing has been conducted in each building, so that the expense of mitigation is avoided if not needed, or to assure that mitigation measures will be sufficient if they are needed. Levels in buildings very close to each other can vary widely. California exposure data for radon are from the late 1980s, thus the exposure assessment needs to be reevaluated which may affect the priority level. The California Department of Health Services (DHS) has established a list of certified providers of radon services, in three categories: testers, mitigators, and laboratories.

## 6.2 MEDIUM RANKED SOURCE CATEGORIES

The pollutant source categories included in Table 6.2 are lower in priority than those above, but nonetheless include sources that warrant mitigation.

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- **Architectural coatings**, such as paints, and lacquers, are available in “low VOC” versions due to formulation changes targeted toward reducing outdoor ozone. However, they are not directly regulated by the state. ARB develops Suggested Control Measures and provides guidance and technical assistance to air quality management districts in the state, 22 of which have adopted rules to reduce VOC emissions from coatings. Like building materials, architectural coatings are widely used and have a high loading in indoor environments when used, due to the large surface areas they typically cover. Additionally, some components of coatings can be harmful, especially if breathed for extended times such as by painters in non-industrial work settings, but are not necessarily addressed through reactive VOC reductions. However, because reductions have been achieved in districts that cover 95% of the California population, this source category is ranked as a medium priority.
- **Consumer products and personal care products** have been regulated by ARB to reduce emissions of reactive VOCs in order to reduce outdoor smog formation. Reactive VOCs, and in some cases toxic air contaminants, have been reduced substantially through reformulation of a number of product categories. In reducing VOC content to comply with ARB regulations, manufacturers often use water-based technologies and use VOC exempt compounds such as acetone. To prevent increased use of TACs, ARB has also prohibited the use of perchloroethylene, methylene chloride, and trichloroethylene in 13 categories. Antiperspirants and deodorants are not allowed to contain any compounds identified as TACs. Also, the Board recently approved a rule to remove *para*-dichlorobenzene from solid air fresheners and toilet/urinal care products.

Depending on the product, zero and ultra-low VOC products are not always commercially and technologically feasible. Despite the breadth of products addressed under ARB’s consumer products regulations, not all types of consumer products are regulated. Products such as wallpaper and vinyl coverings or plastic products may emit chemicals, but are not addressed in ARB regulations. Additionally, due to the nature of some products (household cleansers, air fresheners, stain removers, etc.), the user is in close proximity to the release of chemicals during use, and experiences greater pollutant exposure than individuals who may be elsewhere in the room or building where the product is used. Thus, there is an apparent need to reduce emissions from consumer products to prevent high personal exposures and risks, and to address types of products not currently regulated under ARB’s programs. Because of the ARB’s progress to date with chemically formulated products, some of the highest emitters, this category is ranked medium rather than high.

Chemical reformulations, emission limitations, content limits, and/or product use restrictions of consumer products are mitigation approaches that could result in further significant risk reductions, especially for product users. Such measures have already been taken for many products under ARB’s consumer product authority.

- **Household appliances and office equipment** such as computers, copy machines, and vacuum cleaners can emit a variety of pollutants such as particles, ozone, various VOCs of concern, phthalates, and PBDEs. Emissions from each type of appliance could be addressed through emission limitations and/or local exhaust requirements. Examples of a specification for local exhaust would be the placement of a large copy machine under a strong exhaust ventilation hood, or specifying a level of exhaust ventilation for a room. In most cases, these pollutants are emitted directly into the living or working area, and thus are of concern. Additionally, more and more office equipment is being purchased for use in the home, increasing the number of people potentially exposed in the home environment. Because emissions information on many specific sources in this category is outdated or

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lacking, mitigation efforts for these products are a medium priority. As further research is conducted on these sources, some products may become a high priority for emission reductions.

**Table 6.2 Medium Priority Source Categories for Mitigation<sup>1</sup>**

<b>SOURCES OF POLLUTANTS<sup>2</sup></b> (listed alphabetically)	<b>EXAMPLES OF POLLUTANTS<sup>3</sup> EMITTED</b>	<b>POTENTIAL APPROACH TO MITIGATION<sup>4</sup></b>	<b>DIRECT STATE AUTHORITY TO TAKE IAQ MITIGATION ACTIONS</b>
<b>Architectural Coatings</b> (e.g., paints, sealants, lacquers, varnishes)	Formaldehyde, acetaldehyde, ethylene glycol, metals, others	Emission limitations, chemical reformulations, use restrictions to reduce TACs & nonreactive VOCs with health impacts;	No
<b>Consumer Products</b> (e.g., household and institutional cleaners, furniture- and floor-care products, air fresheners, stain removers, detergents) <b>Personal Care Products</b> (e.g., products used for hair and skin care)	Methylene chloride, para-dichlorobenzene, perchloroethylene, toluene, benzene, naphthalene, formaldehyde, acetaldehyde, metals, others	Emission limitations, chemical reformulations, and product use restrictions to reduce TACs and nonreactive VOCs with health impacts; labeling program	Limited (some indirect)
<b>Household &amp; Office Equipment and Appliances</b> (computers, photocopy machines, vacuum cleaners)	Particles, styrene, some other VOCs, phthalates, ozone, PBDEs	Emission limitations, local exhaust requirements	No
<b>Pesticides</b> (insecticides, herbicides etc, used indoors and outdoors; track-in, drift.)	Chlorpyrifos, diazinon, permethrin, DDT, dieldrin	Formulation and application changes for indoor use	Limited

1. Individual sources may be higher or lower than the source category ranking.
2. All of the examples of pollutant sources may not emit all of the pollutants listed in the corresponding box in column two.
3. Air pollutants may be identified as toxica air contaminants (TACs) by the California Air Resources Board, and/or identified as Proposition 65 chemicals; or, criteria (traditional) air pollutants.
4. Public education, economic incentives, and non-regulatory approaches should also be used where appropriate. The actual approach taken would be determined after extensive discussions with the relevant industries, in consideration of costs, feasibility, and effectiveness.

- **Pesticides** are used indoors and around the perimeter of buildings to control household pests such as ants, spiders, and cockroaches. Pesticide residues may be more persistent indoors than outdoors due to the lack of natural degradation forces such as ultraviolet light, high temperatures, wind, and rain. Pesticides also can be tracked into the home from outdoor application and drift after outdoor spray application. In rural areas, indoor concentrations can be greater due to increased use of pesticides for agricultural purposes.



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Levels of pesticides can be measured in both air samples and house dust samples. Children are often exposed through multiple pathways, via dermal exposure and ingestion from pesticides absorbed onto floor dust particles, in addition to inhalation exposure. Thus, indoor pesticide levels and exposures warrant further attention. Pesticides are ranked in the medium category because programs are available to address them. The U.S. EPA and the California Department of Pesticide Registration (DPR) govern the use of pesticides in California. The U.S. EPA banned the use of chlorpyrifos and diazinon in indoor environments in 2000 and 2001, respectively. Continued research and intervention by the U.S. EPA and DPR are needed to assure that the least toxic pesticides are registered for indoor use and that the formulations prevent excessive human exposure. Most importantly, the implementation of integrated pest management approaches should be expanded to reduce the need for pesticide applications.

## 7. OPTIONS TO MITIGATE INDOOR AIR POLLUTION

This report has shown that there are many sources of indoor air pollution that produce substantial adverse health effects, result in lost productivity, and require considerable expenditures for health care. Despite these facts, there is no systematic program to improve indoor air quality, there are relatively few regulations or standards to specifically address indoor air quality problems, and few resources focused on effectively addressing problems and promoting improvements. Current efforts to address indoor pollution are not commensurate with the scope of the risk to health it poses to Californians.

### 7.1 GENERAL MITIGATION OPTIONS

Ambient (outdoor) air quality is protected through a comprehensive system. In California and under federal law, ambient air quality standards are established for traditional (criteria) pollutants and must be attained. Under other state authority, pollutants identified as toxic air contaminants must be reduced to the maximum extent feasible. The approach used to reduce toxic air contaminants in ambient air, in which source emissions are reduced without setting enforceable air quality levels, seems most applicable to indoor air. Actions to reduce indoor emissions and exposures would assure reduction of exposure and risk from key sources, and should be a major component of a new effort to address indoor air. Other approaches including public education, product testing and labeling, and setting of maximum exposure guideline levels, should also be part of the mitigation program. The following elements of an indoor air pollution reduction program are recommended for consideration:

1. **Create a management system for indoor air quality** that establishes and assigns authority and responsibility for assessing indoor health problems, identifying the actions needed to reduce the most significant problems, and setting guidelines, emissions limits, or other requirements that will be effective in reducing the health impacts of indoor sources. As discussed in Sections 4 and 6 of this report, many agencies' actions affect indoor air quality, and a few have limited authority over some aspect of indoor air quality, but no state (or federal) agency has the authority or mandate to conduct a comprehensive indoor air pollution mitigation program. Such a program is needed, and should be fully coordinated with activities of other agencies whose actions affect indoor air.
2. **Establish emission limits**, when needed, for indoor pollutant sources that pose excessive risks due to their indoor emissions. These might include air cleaners, building materials, furnishings, combustion appliances, and others. While ventilation authority exists in the Energy Commission and Cal/OSHA, no state agency has a direct mandate to establish emission limits for indoor sources for the purpose of reducing indoor exposure and risk. Establishment of such limits would better protect public health, and may reduce (but not eliminate) the amount of ventilation needed under certain circumstances in some buildings, thus saving energy. Compliance could be accomplished by requiring emissions testing through an independent laboratory certified by the state, and submittal of the data to the lead agency.
3. **Require manufacturers to submit building materials, furnishings, combustion appliances, consumer products, and other significant sources for emissions testing** by an independent laboratory certified by the state, and to report those results to the state and to the public. Also, require results to be transmitted to the public via product labeling or accompanying materials in language consumers can understand.

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4. Implementation of a required test program could prove to be an effective approach, at least for reducing indoor pollutant levels in new buildings. A prototype emissions testing program has already been developed for state sustainable building projects: Section 01350 (State of California, 2002) is a testing and assessment protocol developed for reducing VOCs from building materials and furnishings, and is designed to protect human health. A partial list of products that meet this specification is available at <http://ciwmb.ca.gov/GreenBuilding/Specs/EastEnd/>. However, there is currently no requirement for state agencies or others to use these guideline emission specifications, and only limited incentive for them to do so. Other national and international emissions test protocols that are widely used also are available. ARB also has a consumer products test program with test methods (for VOC content) that could be adapted for a broader array of products. Currently, only a few laboratories have the chamber facilities and expertise to conduct such emissions tests. Thus, testing requirements would need to be phased in. However, required testing would prompt other laboratories to obtain appropriate chamber equipment and participate.
5. **Make children's health in schools, homes, and care institutions the top priority.** Implement the recommendations for schools in Section 7.2 of this report. In schools and public daycare centers, require the use of building materials that are certified to be low-emitting. Require that school HVAC systems be quiet (under 45 decibels) and well maintained. Increase efforts to reduce children's exposure to environmental tobacco smoke. Increased education and outreach efforts to smoking parents and caretakers are needed to inform them of the health dangers of second-hand smoke, and the actions they should be taking to protect children under their care from these dangers.
6. **Develop indoor air quality guidelines** for homes, schools, offices, and institutional living quarters. These would largely identify "Best Practices" for the design, construction, operation and maintenance of public, commercial, school, and institutional buildings. In some cases, they might include the identification of healthful levels or "bright lines" for some pollutants to be used as goals for mitigation activities and "best practices", but would not have an associated compliance program. They should also include valid certification requirements for professionals directly involved in indoor air quality-related occupations; performance measures for buildings and appliances; and others. Full commissioning should be required for all new public, commercial, and institutional residential buildings, to assure that they are constructed and operate as intended, and that they provide acceptable indoor air quality. ASHRAE Guidelines (1993, 1996) provide basic guidance for building commissioning, but state requirements are needed.
7. **Amend building codes to address indoor air quality, with a focus on assuring adequate ventilation under all circumstances.** For example, unvented cook stoves, ovens, and combustion appliances should not be allowed in residences. They should be vented to the outdoors, such as through direct venting or an automatic (but quiet) exhaust fan that is activated when the appliance is turned on. Similarly, building codes should be established and enforced to prevent mold problems, residential ventilation issues, and others.
8. **Fund an outreach and education program focused on professionals, including** health professionals, teachers, school facility managers, and others who must be able to identify and remedy indoor air quality problems. Such individuals have many obligations, yet play a key role through their occupation in initial identification, prevention, and mitigation of indoor air quality problems. Most need more in-depth information and training on indoor air quality

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than they typically have had. Training and technical assistance should be provided for the private sector to develop the skills and services needed for high-quality building commissioning, operation, and maintenance.

10. **Conduct more research on indoor air quality.** Several high priority areas are specifically identified in this report for further research. Because of the known serious health impacts of ambient PM and recent studies showing high emissions of PM from indoor sources, research on the health effects of indoor PM are a high priority. The health effects of terpene-ozone reaction products and the extent of people's exposures to them, as well as other indoor chemical reaction products, also are key areas warranting focused research. There are many new chemicals introduced into the product mix each year, yet few have had full health and exposure studies completed. The effects of more recently identified indoor chemicals, such as PBDEs, warrants further investigation. Improved methods and protocols to detect indoor dampness and hidden mold growth are also needed. Synergistic and cumulative health effects are suspected for a number of indoor pollutants with similar structures or properties, yet little research has been conducted in this area. Finally, mitigation approaches assumed to be effective have sometimes been found to be much less effective than anticipated; the effectiveness of recommended or required mitigation measures should be confirmed through appropriately designed studies to assure that the necessary reductions in exposure and risk will be achieved.
11. **Fund an Innovative Clean Air Technology program (ICAT) for indoor air quality** to foster the development and commercialization of legitimate, cost effective technologies that can improve IAQ. For example, improved low-noise ventilation technologies, improved air monitors and assessment tools, and effective low-noise air cleaners are needed. ARB's current ICAT program, focused on improving outdoor air quality through improved technology, has been very successful in bringing new technologies to commercialization in California, adding new options for reducing air pollution while also bringing jobs and investment into the state. An indoor air quality ICAT program would be expected to do the same.

All of these suggested mitigation options are feasible if appropriate mandates and resources are provided. The feasibility of individual measures, such as emission limits for a specific type of product, cannot be determined without substantial additional information. As discussed in previous sections of this report, alternative products or formulations are already available for some of the indoor sources of current concern. However, prior to taking any regulatory action, a more detailed assessment of the specific remedies available, including technological and economic feasibility, would be needed. Additionally, like ARB's current regulatory programs, any emission limitations or other mitigation measures would be developed with continuous discussion and review by stakeholders, the public, and other state agencies.

## 7.2 SOLUTIONS FOR SCHOOLS

The ARB and DHS recently completed a report on a statewide study entitled *Environmental Health Conditions in California's Portable Classrooms* (ARB/DHS, 2004). The study was required by the State Legislature (AB 2872 Shelley; HSC § 39619.6) as a result of concerns regarding reports of mold contamination, inadequate ventilation, elevated levels of volatile chemicals, excessive use of some pesticides, and other problems. The study was funded to help identify the extent of these problems and to determine whether those problems warranted response by the state and/or schools and school districts.

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The study included kindergarten through 12<sup>th</sup> grade public schools. A large, representative sample of both portable and traditional classrooms was studied throughout the state. The results of this comprehensive study have been condensed into a report to the Legislature, and provide important information for state and local decision-makers regarding the degree to which California classrooms provide a safe, healthful, and productive learning environment for children. The report summarizes serious conditions identified in the study that need to be addressed at the State and local levels, and discusses options for improving conditions in both portable and traditional classrooms. The key results and recommendations included in the report are summarized below. The recommendations were developed in consultation with relevant state agencies, industries, school officials, and other interested stakeholders.

### 7.2.1 Problems Identified

The report identifies and addresses a number of environmental problems that were frequently found in classrooms throughout California. These problems were found in both portable (relocatable) and traditional (site-built) classrooms; however, some of the problems were found more frequently in portable classrooms. Government standards and guidelines that are designed to protect children in classrooms and other buildings are essentially lacking. Thus, in this study, results were compared to the most relevant environmental health guidelines and standards available, primarily from professional societies (such as those for ventilation and lighting) and government agencies, such as the Office of Environmental Health Hazard Assessment (OEHHA) and the California Department of Industrial Relations (Cal/OSHA). The primary problems found include:

- Inadequate ventilation with outdoor air during 40% of class hours, and seriously deficient ventilation 10% of the time. This is due largely to teachers turning off HVAC (heating, ventilating, and air-conditioning) systems because of excessive noise and to other factors such as closed outdoor air dampers and inadequate HVAC capacity.
- Temperature and humidity levels outside of professional standards for thermal comfort in about one-fourth of the classrooms.
- Formaldehyde air concentrations above guidelines for preventing acute eye, nose, and throat irritation in about 4% of the classrooms; and, nearly all classrooms exceeded formaldehyde guidelines for preventing long-term health effects, including cancer. Elevated formaldehyde is due primarily to the use of formaldehyde-containing building materials and furnishings.
- Noise levels in all classrooms exceeded the national voluntary acoustic standard for unoccupied classrooms of 35 decibels, a somewhat controversial standard that has not been adopted by any California agency. About one-half of the classrooms also exceeded 55 decibels, the level used by many communities in the state for their outdoor nuisance regulations. Excess noise was due primarily to noisy HVAC systems. Noisy lighting and noise from nearby outdoor activities also contributed. Excess noise directly impacts indoor air quality in the classrooms: when teachers turn off the systems due to noise, classrooms become stuffy and indoor pollutant levels rise.
- Lead, arsenic, and numerous pesticide residues in classroom floor dust; these residues are a concern because they can be inhaled, ingested, or absorbed through the skin (pesticides) by children, especially very young children who sit on the floor and put their hands in their mouths.

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- Obvious mold in about 3% of classrooms; water stains, excess wall moisture, and other potential mold indicators in about one-third of classrooms; musty odors reported by 69% of teachers. These conditions are often attributable to inadequate maintenance.
- Lighting was inadequate in about one-third of the classrooms.

### 7.2.2 Report Recommendations

To address the breadth of problems identified and the many actions needed at all levels to resolve them, a total of 16 recommendations are discussed in the report to the Legislature. These are presented in two groups in the report. Group 1 includes high priority, high benefit actions that can be achieved at relatively low cost and should be accomplished in the near term, while Group 2 recommendations, also a priority, will require a longer timeframe and/or more substantial resources to accomplish. The recommendations fall into four general approaches needed to remedy and prevent the problems seen. The state should:

- Direct and assist schools to comply with state regulations, especially workplace regulations (Cal/OSHA) related to building operation and maintenance.
- Develop and promote “Best Practices” for design, construction, operation, and maintenance of school facilities.
- Improve support (both funding and training) for school facilities and staff.
- Establish guidelines and standards for school environmental health that are protective of children.

The specific Group I recommendations are:

- Schools, districts, and the state should assure that all school buildings meet all relevant state regulations, especially the Cal/OSHA workplace regulations regarding ventilation, sanitation and water intrusion, and illness and injury prevention. Many schools do not meet Cal/OSHA occupational health requirements.
- Schools and school districts should conduct “self-assessments of basic health and safety conditions, similar to the Facility Self-inspection Program undertaken by the Los Angeles Unified School District (LAUSD). Checklists are available on the web.
- The state should require schools to develop indoor environmental quality management plans. The U.S. Environmental Protection Agency’s *IAQ Tools for Schools Program* provides guidance and free kits to accomplish this. The kit is available free of charge at <http://www.U.S.EPA.gov/iaq/schools/>.
- The state should establish a policy to incorporate “Best Practices” into the design, construction, operation, and maintenance of California schools, especially the measures developed by the Collaborative for High Performance Schools (CHPS). The CHPS *Best Practices Manual* at <http://www.chps.net/> provides guidance for measures that will improve schools while also saving energy and reducing long-term costs.

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- State-level review by the Division of the State Architect of the designs for new schools should be expanded to include elements such as ventilation systems and building materials in addition to current elements such as fire and life-safety provisions.
- Portable classrooms (and traditional classrooms) should be sited correctly, away from busy roadways and with proper drainage.
- The state should implement an interim requirement for new classrooms of a maximum noise level of 45 decibels, unoccupied, until a specially-convened task force can determine an appropriate level for California schools (see below).

Group 2 recommendations specify that:

- The state and school districts should assure stable, long-term funding mechanisms and sources for both construction and preventive maintenance; currently, funding fluctuates from year to year, especially for the Deferred Maintenance Program.
- The state should develop and offer focused training programs for school facility managers, custodial staff, and teachers, in cooperation with interested organizations; those closest to the classrooms often are not aware of current “best practices” for operation and maintenance of classrooms. A concerted, ongoing training program could go far to improve conditions in classrooms.
- Integrated Pest Management Programs should be implemented at all schools.
- Older portable classrooms should be retired when they become unserviceable or do not provide an adequate learning environment for children.
- The state and school districts should develop and require full new building commissioning procedures.
- The state should improve its database of school facilities: currently, there is no complete database on the condition, location, or even number, of school buildings.
- The state should convene a task force of experts to develop a California indoor noise guideline or standard for K-12 schools.
- The state should develop chemical exposure guidelines or standards for classrooms that are protective of children and teachers.
- Portable classrooms should be re-designed from the ground up. Several groups are producing new prototypes that use an integrated “whole building” approach; these should be supported through the demonstration phase to evaluate design changes that provide substantive improvements over older portables.

Some actions have already been taken to begin to address these problems; however, they constitute only a first step toward realizing actual improvements in school conditions. Only a small percentage of schools and districts have actively pursued the many tools that are readily available to them to help improve the school environment. The CHPS' *Best Practices Manual*, U.S. EPA's *IAQ Tools for Schools Kits*, and the LAUSD's “Safe School Inspection Guidebook” are all available on the Internet free of charge, yet the number of California schools utilizing

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each of these tools is small. A proactive effort to implement the recommendations of the report is needed.

The complete *Report to the Legislature on Environmental Health Conditions in California's Portable Classrooms* is available at ARB's website at <http://www.arb.ca.gov/research/indoor/pcs/pcs.htm>.



## 8. SUMMARY

Indoor air pollution poses a significant health risk to Californians, in addition to the known risks from outdoor pollution. Indoor pollutant sources emit gases and particles known to trigger asthma attacks and cause cancer, heart and lung disease, and immediate irritant and neurological effects such as eye and throat irritation and headache. Indoor pollution has repeatedly been ranked in the “High Risk” categories in both federal and state comparative risk projects. Some of the known risks in California include:

- ◆ Indoor air pollution includes many asthma triggers. It has been implicated by national scientists as a factor in the serious increase in asthma observed in recent decades.
- ◆ It is estimated that about 230 excess cancers per year may occur in California due to indoor carcinogens from residential and consumer sources, such as formaldehyde. This approaches the 260 estimated excess cancer cases per year from diesel exhaust and equals about two-thirds of the total outdoor cancer burden. Environmental tobacco smoke also adds significant cancer risk. Also, preliminary estimates show that radon gas from certain soils and rock may contribute to about 1500 excess lung cancers per year.
- ◆ Each year, accidental carbon monoxide poisoning from indoor sources causes about 20-26 accidental deaths, hundreds of avoidable emergency room visits, and hundreds to thousands of cases of avoidable illness.
- ◆ Many VOCs, especially formaldehyde, are typically found at elevated levels indoors, and those levels sometimes exceed health-based guideline levels, such as acceptable cancer risk levels.
- ◆ Indoor sources of PM may be partly responsible for the large numbers of premature deaths, hospitalizations, emergency room visits, and increased respiratory disease associated with PM exposures in California each year, and may contribute to these effects beyond the levels quantified in the outdoor epidemiology studies.
- ◆ Biological contaminants can cause communicable disease, hypersensitivity reactions, and even toxic responses, in addition to their widely recognized role as asthma and allergy triggers in indoor environments. Indoor mold problems have increased over the last decade.

Indoor pollutants significantly impact people’s health because there are many indoor sources of pollutants, which often result in elevated indoor concentrations. Additionally, Californians, like others, spend about 87% of their time indoors, on average. Infants and young children spend the most time indoors, most of it in their homes. Several scientists have calculated that indoor emissions are about 1000 times more likely to be breathed than outdoor emissions.

Indoor pollution is estimated to cost California’s economy \$45 billion each year due to medical costs, lost worker productivity, loss of life, and related factors. This estimate is derived from only partial costs of cancer, respiratory disease, cardiovascular disease, and sick building symptoms. This is believed to be an underestimate; the total cost is likely much higher.

Despite the high health and economic costs of indoor pollution, no state or federal agency has explicit authority to regulate indoor sources of pollution to protect building occupants from

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harmful exposures. Indoor pollution remains the only major environmental health problem that does not have the benefit of a focused risk reduction program.

Many actions could be taken to reduce indoor pollution at relatively low cost that would reap large health benefits. Options for mitigation include the development of emission limitations for building materials, furnishings, consumer products, and appliances; improvements in building codes; focused outreach and education programs; improved technologies; and adherence to current “Best Practices” and other guidelines. Substitute products are available for many products and materials that currently emit high levels of pollutants; thus, emission reductions should be achievable in many products with little impact on consumers and homeowners. This would go far to improve indoor air quality in schools and homes, which require a focused effort because little has been done to improve those indoor environments. Also, the most sensitive members of the population—children, the elderly, and the infirm—spend most of their time there. Focused public education, improved appliance standards, and modified building codes would also foster improved indoor air quality in homes and schools. Following current “Best Practices” in design, construction, operation and maintenance is a non-regulatory approach that could yield large gains in indoor health in all types of buildings. Increased effort to publicize and enforce existing Cal/OSHA regulations in schools and non-industrial workplaces would also go far to reduce indoor pollution in those environments; those regulations are in place, but are often not followed.

Perhaps most importantly, a comprehensive management system with responsibility for indoor air quality is needed to combine and coordinate the options indicated above into a cohesive, effective program. That program should include a more detailed prioritization of mitigation actions, careful assessment of the technical and economic feasibility of specific actions, and continued discussion with, and input from, interested stakeholders and members of the public. Such a program also should be closely coordinated with state agencies whose programs include some existing authority related to indoor air quality, especially Cal/OSHA and the Energy Commission, to assure consistency and avoid duplication of effort

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## 10. GLOSSARY

<b><u>TERM</u></b>	<b><u>DEFINITION</u></b>
Active/Passive Sampling	Active sampling depends on a mechanical process like pumping to collect the sample at a known rate; this is used for VOC and aldehyde sample collection. Passive sampling involves non-mechanical processes, usually diffusion, in which the air is sampled at whatever rate it passes across a badge surface or tube opening.
Air Changes per Hour	ACH, the volume of air moved in one hour. One air change per hour in a room, home, or building means that the equivalent of the volume of air in that space will be replaced in one hour.
Air Cleaners	These are devices designed to remove pollutants from a room. Air cleaners can be portable, or part of a central air system. Air cleaners can be mechanical, employing a filter to remove pollutants, or electronic using a small electrical charge to collect particles from air pulled through a device.
Air Fresheners	These devices are promoted to neutralize odors rather than remove pollutants. Products often emit a fragrance which diffuses into the air. Some styles can spray fragrances and be programmed for fragrance dispersion.
Air Flow Rate	The rate at which air moves into a space. Expressed in units of air changes per hour or cubic feet per minute.
Air Handling Unit	HVAC (heating, ventilation and air conditioning) unit. Refers to equipment that includes a blower or fan, heating and/or cooling coils, and related equipment such as controls, condensate drain pans, and air filters. Does not include ductwork, registers, or grilles, or boilers and chillers.
Allergen	A chemical or biological substance (e.g., pollen, animal dander, or house dust mite proteins) that induces an allergic response, characterized by hypersensitivity.
Ambient Air Quality Standards (AAQS)	State (ARB) and federal (U.S. EPA) enforceable regulations designed to protect the public from the harmful effects of traditional pollutants in outdoor air.
Asthma	A chronic disease of lung tissue which involves inflamed airways, breathing difficulty, and an increased sensitivity to allergens and contaminants in the air.
Biological Contaminants	Agents derived from or that are living organisms (e.g., viruses, bacteria, fungi, and mammal, arthropod, and bird antigens) that can be inhaled and can cause many types of health effects

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	including allergic reactions, respiratory disorders, hypersensitivity diseases, and infectious diseases. Also referred to as biological agents.
Comfort Measures	Factors that determine human perception of thermal comfort, including temperature, relative humidity, and draft
Commissioning	Testing the important building systems before occupancy to ensure that they operate the way the designers expect and that they serve the needs of the building occupants. Systems that can be tested include HVAC, electrical, energy management, plumbing, etc.
Fungi	A group of organisms that lack chlorophyll, including molds, mildews, yeasts, mushrooms.
Integrated Pest Management (IPM)	A pest management strategy that focuses on long-term prevention or suppression of pest problems through a combination of techniques such as monitoring for pest presence and establishing treatment threshold levels, using non-chemical practices to make the habitat less conducive to pest development, improving sanitation, and employing mechanical and physical controls. Pesticides that pose the least possible hazard and are effective in a manner that minimizes risks to people, property, and the environment, are used only after careful monitoring indicates they are needed according to pre-established guidelines and treatment thresholds.
Micron	A unit of length equal to one millionth of a meter; a micrometer.
Microorganism	A microscopic organism, usually a bacterium, fungus, or protozoan.
Natural Ventilation	The movement of outdoor air into a space through intentionally provided openings, such as windows and doors, or through non-mechanical ventilators, by wind, air pressure differences, or other natural, non-mechanical means.
Permissible Exposure Limits (PELs)	Enforceable pollutant exposure limits determined by OSHA that are designed to protect healthy adult workers in industrial environments from adverse health effects associated with pollutant exposure. None of these limits are targeted toward protecting children.
Pesticides	A pesticide is any substance or mixture of substances intended to prevent, destroy, repel, or mitigate any pest. Though often misunderstood to refer only to insecticides, the term pesticide also applies to herbicides, fungicides, disinfectants, and antimicrobials. Under U. S. law, a pesticide is also any substance or mixture of substances intended for use as a plant regulator, defoliant, or desiccant.

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Polycyclic Aromatic Hydrocarbons (PAHs)	A class of stable organic molecules comprised of only carbon and hydrogen. They are a common product of combustion from automobiles, airplanes, woodburning, cigarettes, and some types of cooking. Many of these molecules are highly carcinogenic and very common.
Portable Classrooms	Classrooms that are designed and constructed to be moveable and transportable over public streets, also known as temporary or relocatable classrooms.
Quality Control (QC)	Internal checks on the operation of sample collection and/or sample analysis. Methods for determining the operation include blanks, spiked samples, flow checks, and duplicate samples. QC measures can be used to determine accuracy, bias, and precision of the data reported.
Real-time Monitoring	This type of environmental measurement gives instantaneous information at the point of sampling; measurements are recorded as often as every minute, every second, or in fractions of a second.
Reference Exposure Level (REL)	The concentration level at or below which no adverse health effects are anticipated for a specified exposure duration. RELs are based on the most relevant, adverse health effect reported in the medical and toxicological literature for the population group known to be most sensitive to the chemical. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Since margins of safety are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact will occur. OEHHA provides acute (1-hour) and chronic (lifetime, non-cancer), RELs for a number of chemicals, and has developed an 8-hour "indoor" REL for formaldehyde.
Relative Humidity	The measure of moisture in the atmosphere, expressed as a percent of the maximum moisture the air can hold at a given temperature.
Return Air	Air removed from a space by the HVAC system to be recirculated or exhausted.
Sick Building Syndrome	A set of symptoms (including headache, fatigue, and eye irritation) typically affecting workers in modern airtight office buildings, believed to be caused by indoor pollutants (such as formaldehyde fumes or microorganisms).
Supply Air	Air delivered to the conditioned space by the HVAC system and used for ventilation, heating, cooling, humidification, or dehumidification. It is usually a combination of outdoor air and return air.

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Traditional Classrooms	Classrooms in permanent, site-built school buildings.
Variable Air Volume System	Air handling system that conditions the air to a temperature using a varying amount of outside airflow based essentially on the outdoor temperature.
Ventilation	The process of intentionally supplying and removing air by natural or mechanical means to and from any space.
Volatile Organic Compounds (VOCs)	Compounds that evaporate quickly from the many housekeeping, maintenance, and building products made with organic chemicals. These compounds are released from products that are being used and that are in storage. Many are carcinogenic, neurotoxins, or mucous membrane irritants.